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EXPERIMENTS ON THE ELECTRICAL EXCITATION OF THE VISUAL AREA OF THE CEREBRAL CORTEX IN THE MONKEY.

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THE visual area of the cerebral cortex in the monkey, so far as it is determinable from the results of extirpation and of electrical excitation, comprises the whole of the occipital lobe. It perhaps includes a part or the whole of the angular gyrus, although the results of extirpation would appear to show, that the part played by the latter convolution is comparatively small. Removal of both occipital lobes produces total and permanent blindness, whereas destruction of the cortex of both angular gyri is not followed by any appreciable permanent defect of vision.[1] But, as will presently appear, electrical excitation of the posterior limb of the angular gyrus produces movements of the eyes similar to those which are produced by

[1] H. Munk, 'Verhandlungen der Berliner physiol. Gesellschaft,' 1878; and Schäfer, 'BRAIN,' January 1888. Of two monkeys in which, in conjunction with Dr. Sanger Brown, I completely removed both occipital lobes, leaving the angular gyri intact, one was totally blind and the other nearly so, having possession, however, of a small part of the visual field, so as to be able to see objects held well above the horizontal visual plane. On post-mortem examination it proved that the lesion did not in this case involve quite as much of the under-surface of the hemispheres as in the other. This agrees with the results of the excitation-experiments herein to be described, from which it will appear that stimulation of the under-surface of the posterior part of the hemispheres produces an upward inclination of the visual axes.

excitation of the adjoining portion of the occipital lobe; it is probably correct, therefore, to include at least this portion of the angular gyrus in the visual area.[1]

The results which previous observers have obtained from excitation of the visual area as thus defined are contradictory. Ferrier obtained movements of the eyes to the opposite side with closure of the lid-openings, and contraction of the pupils, from excitation of the whole of the angular gyrus, the eyes being usually directed upwards when the electrodes were applied to the anterior limb, downwards when applied to the posterior limb. He "never obtained any reactions on stimulation of the occipital lobes."[2] Luciani and Tamburini got only the conjugate deviation to the opposite side without any constant upward or downward inclination, and they also obtained similar movements, but of a less marked character, on excitation of the convexity of the occipital lobe.[3]

I have carefully explored these parts of the hemisphere with the induced interrupted current in a number of monkeys, and have obtained the following results:[4]

[1] A similar result is obtained on stimulation of the upper end of the superior temporal gyrus, and of the adjacent portion of the middle temporal. This may be due, as is usually supposed, to the calling up of subjective auditory sensations; but the possibility of this part also belonging to the visual area, with which it is anatomically continuous, must not be overlooked.

Whilst engaged in enumerating the parts, excitation of which produces movements of the eyeballs, it is right that the frontal area for movements of the head and eyes should be included in the list. But this is evidently different (more direct) in its mode of action, for its removal causes paralysis of those movements (Ferrier), and moreover, as I have elsewhere shown ('Proc. Roy. Soc.,' 1888, and 'International Journal of Anatomy and Physiology,' 1888), its period of latency is much shorter than that of the posterior (visual and auditory ?) areas.

[2] Ferrier, 'Functions of the Brain,' second edition, p. 244.

[3] Luciani and Tamburini, 'Sulle funzioni del cervello. Centri psico-sensori corticali.' Reggio-Emilia, 1879. (Abstract in 'BRAIN,' Vol. II.)

[4] For investigating the external surface, platinum-pointed electrodes of the ordinary pattern used in physiological experiments were employed; but for the exploration of the mesial and under surface, electrodes of a special pattern were constructed. These had their wires guarded, except on one side near the extremity, by paraffined paper, and were of such a form that they could be easily slipped between the brain and dura mater, and the excitation conducted through the unguarded parts of the wire directly to that part of the concealed surface which it was desired to stimulate.

A Du Bois-Reymond coil, with vibrating hammer, worked by a Daniell cell and with Helmholtz's side-wire inserted in the primary circuit, was used throughout. Ether was employed as an anæsthetic.

Excitation of the posterior limb of the angular gyrus, the upper end of the middle temporal gyrus (into which it is continued), and of the whole of the cortex of the occipital lobe, including its mesial and under-surfaces, produces conjugate deviation of the eyes to the opposite side. The excitable area also includes the part of the mesial surface immediately in front of the internal parieto-occipital fissure (quadrate lobule). The movement produced is either a purely lateral one, or a lateral movement combined with either a downward or an upward deviation, according to the part to which the electrodes are applied.

1. When they are placed upon an area which includes the posterior limb of the angular gyrus, the part of the second temporal gyrus with which it is immediately continuous, the portion of the external surface of the occipital lobe immediately behind the external parieto-occipital fissure, and the anterior and upper part of the mesial surface immediately in front of (quadrate lobule) and immediately behind the internal parieto-occipital fissure, the deviation is accompanied by a downward inclination, which is relatively most marked when the electrodes are applied on or near the mesial surface.

2. When they are placed upon an area which includes the posterior extremity of the lobe, its tentorial surface, and the posterior and lowermost part of its mesial surface, the deviation is accompanied by an upward inclination, which is also relatively most marked on applying the stimulation on or near the mesial surface.

3. When the electrodes are applied to an intermediate area, which is chiefly formed by the remainder of the convex external surface of the lobe, but also extends over the inner border to the mesial surface, where it becomes greatly narrowed, the deviation is simply lateral.

The results, therefore, of electrical excitation of this posterior (or visual) region of the hemisphere indicate a division into three zones; not sharply marked off from, but merging into one another. The first, or uppermost zone, is connected with downward inclination of the eyes; the second, or lowermost, with upward inclination; and the third, or intermediate zone, which is wide externally and narrow mesially, with a simple

lateral movement. But a lateral movement is also produced along with the downward or upward inclination by excitation of the upper and lower zones, although the vertical movement of the globes may be so powerful as almost to obscure the lateral inclination. This most frequently happens when the excitation is applied to the mesial parts of the upper and lower zones. Usually the upward direction of the visual axes, consequent upon stimulation of the lower zone, is accompanied by elevation of the upper eyelid, and the downward direction of the visual axes, consequent upon stimulation of the upper zone, by depression of the upper eyelid; but these effects have not been constant in my experiments, nor have the changes in the pupils which are sometimes observed accompanying the movements of the eyes.

I have not obtained any effect from stimulating, *with weak faradization*, the posterior limb of the angular gyrus, even when well-marked results have been obtained, with a strength of current barely perceptible to the tongue, from the parts which have just been considered.

I am unable to say exactly how far forward on the under-surface of the hemisphere these effects of excitation of the cortex may extend. The conjugate deviation to the opposite side, combined with upward inclination, is produced certainly as far as what is anatomically described as the limit of the occipital lobe on the under surface, and indeed the excitable region appeared to extend in advance of this limit; but it was not easy to say in experimenting upon this (concealed) part, to what spot precisely the electrodes had been applied.

I have occasionally obtained from stimulation on or near the quadrate lobule marked contraction of the pupils, such as is produced when a strong light is thrown directly into the eye. More usually, however, the movements, whatever the part stimulated, are accompanied by dilatation of the pupil.

So far as I have been able to observe, the visual axes always maintain parallelism when one side only of the hemisphere is stimulated. On one occasion I stimulated various corresponding points of the two hemispheres simultaneously. The result was usually a struggle between the muscles producing lateral movement, and the eyes either quivered without

moving to one side or the other, or the one set of muscles preponderated over the other, so that a lateral movement to one side resulted. In one case, when the electrodes were placed on corresponding points of the mesial surfaces, there was slight convergence of the visual axes; but this result was not sufficiently marked or constant to permit much stress to be laid upon it.

If we assume that these various movements of the eyeballs are the result or concomitants of subjective visual sensations set up by the excitation, and the movements take the directions towards which those sensations are projected, then the above experiments indicate a certain connection between the parts of the cerebral visual area and of the retinæ. This connection may be stated as follows:—

1. The whole of the visual area of one hemisphere is connected with the corresponding lateral half of both retinæ.

2. The upper zone of the visual area of one hemisphere is connected with the upper part of the corresponding lateral half of both retinæ.

The lower zone of the visual area is connected with the lower part of the corresponding lateral half of both retinæ.

4. The intermediate zone of the visual area is connected with the middle part of the corresponding lateral half of both retinæ.

We may represent these relations diagrammatically, by supposing the visual areas of both hemispheres to be united at the great longitudinal fissure, and imagining each retina to be expanded and projected in its natural position upon the united area. If this is done, it is at once seen that the identical points of the two retinæ correspond with one and the same spot of the cerebral cortex; that the upper half of each retina falls on the upper part of the united area, and the lower half of each retina on the lower part of that area; that the inner half of the one retina and the outer half of the other each fall upon the same hemisphere, and that a vertical zone, including the centres of both retinæ, corresponds with the mesial part of the united area. This would connect the parts of the retinæ which are concerned in direct or central vision with a part of the mesial surface of the occipital lobes, probably

including a portion of the quadrate lobules. This scheme of connection between the retinæ and occipital lobes differs in several points from the well-known scheme formulated by H. Munk,[1] as the result of extirpation experiments, chiefly upon dogs, but partly upon monkeys. The most important differences relate to the extent of the visual area, and to the part of the occipital cortex which is connected with the central parta of the retina (macula lutea). The visual area (*Sehsphäre*) of Munk is *confined to* the occipital lobes, and the maculæ luteæ are *connected with the middle of the convexity of those lobes.*[2] Consequently, if the middle of the convexity of both occipital lobes is removed, central vision should be abolished. But, contrary to Munk, I have not found this to be the case in monkeys. Indeed, it was the smallness of the effect produced by this bilateral lesion which first led me to doubt the correctness of Munk's scheme. On the other hand, in one experiment which I have performed since obtaining the above results from stimulation of the cortex, there was a marked defect of central vision produced by a bilateral injury of both mesial surfaces. Complete and permanent abolition of central vision did not result, but it was found, post-mortem, that the lesion did not involve as much of the mesial surface of the visual area, especially upon one side, as was intended at the operation. It will be necessary to follow out the question more closely by the extirpation method, and this I intend in the near future to attempt; but the difficulty of arriving at reliable conclusions from localised extirpations of the visual area is enormous. For the animals soon acquire the habit of compensating for local defects in the visual field, by rapid movements of the eyes, so as to baffle all attempts to determine the existence of such defects. I believe, indeed, that to arrive at detailed conclusions we must await the results of perimetric observations in cases of cerebral lesion in the human subject, and that these experiments upon the visual area of the monkey are chiefly of value as affording indications of the kind of result which is to be looked for in man.

[1] H. Munk, 'Ueber die Functionen des Grosshirns,' Gesammelte Mittheilungen, Berlin. 1881, 5te Mittheilung.

[2] Op. cit. p. 127.

SCHÄFER ON THE TEMPORAL AND OCCIPITAL LOBES.

BY DAVID FERRIER, M.D., F.R.S.

THE opponents of cerebral localisation—a race well-nigh extinct—make capital out of the fact, that the advocates of this doctrine are not agreed among themselves as to the exact extent of the areas or regions supposed to be endowed with specific functions. They will therefore welcome with satisfaction the contribution to the physiology of the cerebral hemispheres, by Professor Schäfer, in the last issue of this Journal.[1]

In this paper, Professor Schäfer not only differs in important respects from all who have preceded him in this field of enquiry, but exhibits the curious spectacle of one who believes in the rigid localisation of a visual sphere, a centre for common or tactile sensibility, and differentiated motor centres, and yet contends that the important senses of hearing, taste, and smell have, apparently, no local habitation in the brain. For he can obtain no evidence of impairment even, not to mention abolition, of any of these faculties on destruction of any portion of the cortex. And in particular, he asserts, that the temporal lobe—in which it is now very commonly believed that these faculties are centralised, and where alone it would seem they can be, if not diffused among regions already otherwise occupied—may be completely removed on one or both sides, leaving hearing, taste, and smell absolutely intact.

While he thus abounds in negations, Professor Schäfer, who has done some good work in cerebral localisation, gives us no clue to the situation of the centres whose supposed seat he so

[1] "Experiments on Special Sense Localisations in the Cortex Cerebri of the Monkey."—'BRAIN,' Jan. 1888.

ruthlessly demolishes; and he is unable to indicate any use of the temporal lobe, except that its removal on both sides is in some way the cause of a temporary idiocy.

To one who has accepted the principle of localisation in respect to vision, tactile sensibility, and special motor faculties, this should appear a most lame and impotent conclusion.

It is difficult to believe, that Professor Schäfer can have formulated to himself the logical consequences of the position he has taken up, without having had serious mistrust raised in his own mind as to the completeness of his lesions, or the reliability of his methods and observations. But if he has, his faith in his methods must be of an extremely robust character, if it can have impelled him to assume such an equivocal position, and oppose himself to the combined evidence of comparative anatomy, clinical pathology, and actual experimental demonstration.

As I have, in my work on 'The Functions of the Brain,' (page 310) attributed to Professor Schäfer (and his former colleague, Professor Horsley) opinions respecting the auditory centre altogether at variance with those which he now maintains, it is necessary that I should, in the first place, endeavour to clear myself of the charge of having misrepresented him.[1]

In a letter dated March 19th, 1885, Professor Schäfer wrote to me as follows: "We have some monkeys just now which you ought to see. The whole of the temporo-sphenoidal lobe, including the hippocampus, has been removed in them, and we cannot detect any paresis of sensation (except auditory)."

I did not, however, doubt the accuracy of my own observations, and, at my request, Professor Schäfer and his colleague consented to perform the experiment under my supervision and direction. When this was carried out, I had no difficulty in demonstrating to their satisfaction that the lesion had caused profound impairment of common and tactile sensibility on the opposite side of the body. And I had the further pleasure of suggesting to them, and seeing performed, experiments which I had myself planned, viz. the extension of the lesions into

[1] A charge which I observe is made in the 'Phil. Trans.,' 1888, B. 20, p. 20, footnote.

the gyrus fornicatus, or anterior division of the limbic lobe. The results of these experiments demonstrated the physiological unity of the callosal and hippocampal gyri, and confirmed the unity which Broca had indicated on anatomical grounds.

I naturally gathered from Professor Schäfer's letter that he had obtained evidence of the existence of an auditory centre in the temporal lobe. For he expressly excepted auditory from the negative effects as regards other forms of sensibility.

And as he had informed me that—in harmony with my own experiments—he had obtained no indications of impairment of hearing on destruction of the rest of the temporal lobe, apart from the superior convolution, I considered myself justified in quoting his results as confirmatory of my own. Professor Schäfer is of course entitled—nay bound—to change his views, when the evidence on which he based them appears on further consideration to be insufficient; but I do not think that I can be said to have seriously misrepresented them as they at that time stood.

Professors Schäfer and Horsley had given me an opportunity of testing, and I am happy to say of correcting, conclusions which they were inclined to draw adverse to my own. But the first intimation I had of Professor Schäfer's further investigations (in conjunction with Dr. Brown) was the announcement at a meeting of the Physiological Society on February 12th, 1887, of the views which he had already then arrived at, and now puts forward in a more elaborate form. These were first published in the 'Proceedings of the Physiological Society,' No. 2, 1887. I criticised them at the time, and at a subsequent meeting of the Neurological Society, I strongly deprecated this premature publication of conclusions while the evidence was incomplete—seeing that the animals were still alive, and the actual lesions undetermined—as being likely to do harm and prejudice dispassionate enquiry. And I particularly called attention to—and here emphasise—the difficulties that have to be encountered, and the fallacies that are apt to be committed, in investigating the sense of hearing in the lower animals. Care must be taken to discriminate between the sense of hearing proper, and mere reflex reaction to sudden,

sharp, or loud sounds, a reaction not incompatible with real deafness. Mere coincidences in animals keenly on the alert to everything going on around them, must be scrupulously eliminated. The tests of hearing should be such as to entirely avoid attracting the animal's attention by other channels of perception, such as sight, imitation of other animals, a sense of proximity conditioned by vibration, agitation of the air, and such like. The animal's present behaviour should be compared with its past, or with that of normal monkeys under conditions of variation in sound, and special kinds of sound, only, all other things remaining the same. And even then, it often requires long and laborious investigation to eliminate all sources of fallacy. Professor Schäfer is evidently satisfied that he has avoided all sources of fallacy himself, and he quotes in support of his conclusions the opinions of a Committee of the Neurological Society in favour of the retention of hearing in two of his experimental animals, in one of which he says he had scooped out the superior temporal convolution on both sides, "so that not a trace of the convolution in question should remain" (Fig. 5); and in the other he had removed not only the superior temporal convolution, but even the whole temporal lobe on both sides (p. 373).[1]

But Professor Schäfer's figures (Figs. 5 and 6) of the brains of these monkeys must be regarded more as a sort of pictorial representation of his own idea, or wish, as to the extent of the lesions, rather than of the reality. Having myself examined both preparations, I most emphatically deny the accuracy of Professor Schäfer's diagrams. In neither the one nor the other were the superior temporal gyri completely destroyed:—considerable portions remaining absolutely intact in both hemispheres, and notably so in the brain which Fig. 6 professes to

[1] Professor Schäfer has committed a slight irregularity in quoting the opinions of a Committee, before the members of the said Committee had met together, or formulated any report of their proceedings.

In the absence of any information as to methods and tests employed by the visitors appointed by the Society, I am willing, without criticism, to attach the utmost weight to the opinions at which they arrived. But of this I have no doubt whatever, that if the evidence of hearing in the animals submitted to their investigation was indubitable, the explanation is to be found in the fact, that the superior temporal gyri were not completely removed in any one of them.

represent. And considering how extensive lesions may be in any given centre—witness the visual centre—without total annihilation of its functions, nothing short of absolute destruction, primarily or secondarily, is likely to abolish all vestiges of the sense to which it is related.

But apart from Professor Schäfer's experiments, let us proceed to consider the question of the locality of the sense of hearing on broader grounds. The evidences of the localisation of the centre of hearing in the temporal lobe—and according to my view, in the superior convolution of this lobe —are as follows: First, the reaction on excitation by the electrical stimulus—a reaction which afforded me the suggestive indication, leading to the verification by destructive lesions, of the relation between this convolution and the faculty of hearing. Electrical irritation of the upper two-thirds of the superior temporal convolution causes a quick retraction or pricking of the opposite ear, as if towards the supposed origin of the sound, combined generally with turning of the head and eyes to that side, and dilatation of the pupils. I have used the term "pricking" as synonymous with retraction and pointing of the auricle in the direction to which sound would naturally be referred. I have nowhere described the reaction as a "pricking *forward* of the ear," such as Professor Schäfer attributes to me (p. 373). The reaction is precisely that which an animal makes when a shrill sound is made towards one side, as may be easily verified in any monkey, or such animal as the cat, whose ears, when at rest, are pointed and directed forward. In the lop-eared rabbit, however, irritation of the homologous region causes sudden elevation of the ear, as well as retraction and exposure of the mouth of the auricle towards the referred origin of the sound. Professor Schäfer says that excitation of very various parts of the cortex (frontal region, middle temporal gyrus, angular gyrus, and occipital lobe) produces "almost precisely the same effects" (p. 372). As I was myself the first to point out the various positions from which movements of the head and eyes to the opposite side might be stimulated, it is scarcely likely that I should describe the movements of the head and eyes as a reaction peculiar to the superior temporal convolution. The special

feature of this reaction is the sudden retraction of the ear which occurs on irritation here, and here only. Professor Schäfer's ingenuousness is not very apparent when he endeavours to conceal the significant differences between the reaction of the superior temporal convolution and that of the other regions above enumerated, under the words "*almost* precisely the same effects."

Next, as regards the evidence from destructive lesions, as supplied by my own experiments, and those which I made in conjunction with Professor Yeo. This consists of an extensive series of experiments reported in detail in the 'Philosophical Transactions,' Part 2, 1875, and Part 2, 1884, involving divers lesions of the temporal lobes;—*negative* as regards affections of hearing when the superior temporal convolution was intact, and *positive* when this was destroyed unilaterally and bilaterally; and all mutually corroborative of each other. No greater importance is attached to these experiments individually than as links in a chain leading up to the conclusive demonstration which was given before the Physiological section of the International Medical Congress in 1881.

In one of the series in which the superior temporal convolution was destroyed on both sides in the upper two-thirds of its extent (the left angular gyrus having been destroyed previously but without permanent effects), the following notes were made as to the condition of the animal on the day after the operation.[1]

"*April 8th*, 10 A.M.—Animal alive and active, and jumped out of its cage when the door was open. Sight was good, and tactile sensation unimpaired. Various experiments were made to ascertain the existence or not of hearing; but it was difficult to devise a test, as the animal was continually on the alert; and it was not easy to make a sound without in any way attracting its attention by sight. The following method was tried: While the animal was sitting quietly by the fire, I retired to the other room, and while watching through the chink of the half-shut door, called loudly, whistled, knocked on the door, tinkled glass, &c., without ever causing it to look round, or give any sign of having heard. I then cautiously approached the animal, and not till it saw me did

[1] See Experiment 15, 'Phil. Trans.,' vol. 165, part 2, 1875.

it give any sign of consciousness of my presence. When the same experiment was repeated, while the monkey and its companion were quietly seated by the fire enjoying the heat, it gave no sign of hearing; while its companion started with alarm, and came with curiosity to ascertain the cause of the sound. . . . 8 P.M.—In the presence of Dr. Burdon Sanderson I repeated the various tests with the view of eliciting signs of hearing. To all it remained without response. It seemed unconscious of my presence when speaking close to its ear, and only started when it caught sight of me."

The last of the series confirming the presence of, and establishing the permanency of, similar symptoms was the now somewhat famous monkey exhibited at the International Medical Congress. In view of certain questions that have been raised as to the exact condition of this animal, I give here verbatim the rough notes written down from day to day.

The subject of the experiment was an unusually active and intelligent monkey, in full possession of all its faculties.

It was designated Monkey F. The extent of the lesions, superficial and deep, in this animal are faithfully represented by photography on Plate 22, Figures 23–38.[1]

"*June 23rd* (24 hours after the operation).—F. to-day is rather dull and drowsy-looking. Some œdema of face and eyelids. Right eye partially closed, the left open. Put out its hand and touched companion sitting near it and eating a piece of bread. It gives distinct signs of perception of touch on hands, feet, ears; moves about perfectly freely. Pays no attention whatever to the loudest sounds which startle the other monkeys, or to whistling, tapping, &c., close to its ears. Oil of bergamot placed on a rod and held at its nostrils caused it to sniff apparently and make movements with its lips. Acetic acid causes it to retract its head sharply, and sneeze or snuff out. Acetic acid to lips causes licking movements of tongue; so also common salt placed on lips. Sees perfectly well, and goes about from one cage to the other through small door.

"*June 25th.*—F. is very bright and active; runs about actively climbs up bars of cage, and peers eagerly through bars at what is going on. Takes things offered it. Took a piece of date, smelt it and threw it down; so also a piece of bread. Took next a piece of cold boiled potato, smelt it, rubbed it in its hands, and began to

[1] 'Phil. Trans.,' p. 2, 1884.

eat with relish. Pays no attention to sounds—tapping, scratching, &c.—on side of cage, which alarm its companion toward either side. This was carried on repeatedly for the space of an hour without eliciting any sign of perception. Going down to the place where the animals are kept, sets all the others on the *qui vive*. No sign of this on the part of F. But as soon as I come within sight, it looks interested and comes to the bars and looks up.

"*June 27th.*—Examined F. with Dr. Lauder Brunton. Tried various tests, sounds, whistling, &c. Could not decide positively. Animal occasionally turned its head, but whether coincidence or hearing, doubtful. At any rate, conclusion arrived at that if the animal not absolutely deaf, its hearing was profoundly affected. Noted that the ears did not twitch as in the case of the companion monkey; nor did it show alarm at sounds.

"*June 28th.*—F. placed in a separate compartment. Did not reply to plaintive cries of its companion. F. tried constantly to see its companion through small apertures in cage. Loud sounds, which made all the others start and look frightened, caused generally not the slightest sign on the part of F. Occasionally it turned its head coincidently with the noise, but whether hearing, very problematical, as at other times no such sign. The ears did not twitch; nor any nervous wincing as in the case of the companion. No response to cries or calling by name. Sight, motor power, and tactile sensibility perfect to all tests.

"*June 30th.*—Repeated the tests with the same result as formerly. The animal is mostly totally unconcerned when its companion is in abject terror, shrinking, winking, and twitching its ears. Every now and then, doubt seems raised by the animal looking at the same time as a noise is made.

"*July 2nd.*—Again examined the animal carefully with Dr. Yeo. In response to various tests—sounds of various kinds—there was most commonly no indication of perception, such as the companion monkey showed by shrinking, grimaces of terror, cries, and twitching of the ears. Occasionally the animal would turn its head coincidently with the sound. But this is only if a sound made towards stairs leading down to cage, where it in general directed its attention spontaneously. *Conclusion:* if not absolutely deaf, hearing very doubtful, and at least no impairment of the other powers of the animal.

"*July 5th.*—Made several experiments with electric bell placed against side of cage. On ringing it, the companion was at first very anxious about the sound, while F. paid no attention, but after a time it ceased to regard it. Cannot yet be sure whether the animal hears at all, but certainly is not at all disturbed by

sounds which make the others start, twitch ears, wink, &c. No twitching of the ear in F. Otherwise perfectly well. Runs about; is very expert in catching flies, which it watches carefully and pursues skilfully. Very much on the alert to all that goes on, and occasionally utters a grunt if it sees anything like water, food, &c., which it wants. Never, however, replies by grunts to those of its companions, and is generally perfectly silent. Formerly it was continually in the habit of grunting, &c., in reply to name, &c.

"*July 7th.*—Have been making numerous experiments, also Dr. Yeo, as to whether the animal hears the noise of a percussion-cap exploded near. It is most difficult to judge. Occasionally it seems to start coincidently with the sound, and yet at other times gives not the slightest sign, while the others invariably start. The dressings removed to-day; all aseptic; wound perfectly healed. Animal well and vigorous, very much on the alert. The ears are now mobile. It moves them during its actions of attention, but I have not yet been able to ascertain definitely whether they twitch to sound. This afternoon, at six, made an interesting experiment while sitting in the cage with its companion (G.), and both quite still, and engaged in examining each other. I made repeatedly a scraping sound on floor with foot as if going up to cage. The companion G. invariably opened its eyes and looked up, whereas there was never any sign on the part of F.

"*July 13th.*—Examination to-day confirms former remarks. Once very clearly percussion-cap excited G., and met with no sign of response from F. Does not answer calls like G., and pays no attention to sounds which cause G. to look up.

"*July 15th.*—Well and active. Gives no signs of recognition to cries, and frequently not the slightest sign of movement, or attention to shots. It is more than doubtful whether there is any reaction at all.

"*July 20th.*—Condition unchanged.

"*July 27th.*—Owing to the death of G., F., which has been disconsolate, and occasionally uttering plaintive cries, was transferred yesterday to the cage of C. (hemiplegic). The two seemed to agree tolerably well, with the exception of squabbles as to food—C. being very indignant, and occasionally attacking F. for seizing the food first. Made various experiments as to hearing, and it seems that the signs of reaction (which have caused so much doubt) to sounds are less evident. The electric bell was transferred to the side of its cage. Ringing it suddenly, caused C. to be very much disturbed and annoyed, whereas F. paid no attention whatever. Coming downstairs to the place where the animals are kept, I observed C. looking out attentively, but F.

remained in back of cage, not coming forward until such signs on the part of C. as to attract notice. Repeated percussion-cap experiments several times. C. invariably started, but to-day I failed to detect any unequivocal indication of this on the part of F. As to other sounds—calling, whistling, &c.—F. pays no attention whatever, whereas the attention of the other monkeys is at once excited. The animal is excessively on the alert, and it follows every movement, and is with difficulty surprised.

"*July 29th.*—The percussion-cap experiment, repeated to-day several times in the presence of Dr. Yeo and J. G. Romanes, was every time negative, causing no reaction or starting, while its companion was invariably much startled and annoyed.

"*August 4th.*—The animal was exhibited to-day before the Physiological section of the International Medical Congress. A percussion-cap was exploded close to it several times before the audience, and it gave no sign whatever of starting; whereas C. started very emphatically.

"*December 12th.*—The animal has kept in excellent health, and is most lively, and continually engaged in active gymnastics. Many experiments were made with the view of testing its hearing. Sometimes it appears to start when a pistol-cap is exploded, so that occasionally it is judged to hear. To-day tried the experiment of calling from the top of stairs. The other monkeys [2] are both on the alert, looking eagerly and making sounds of expectation; but F. pays no attention whatever, and goes on with its own occupation quite unheeding. It does not pay any attention to scratching on cage as the others do, and in general does not respond to sound-calls, &c., which always excite the attention of the others. It does not utter sounds in reply to calls. Occasionally, when left all alone, it utters a short cry of loneliness.

"*March 31st.*—While sitting to-day having its back scratched, a pistol-cap was exploded close to cage; not the slightest start, though the others and Mr. N., who was scratching the monkey, made a sudden start.

"*July 27th.*—The animal has remained well and lively without showing any new symptom, and still to all appearance exactly in the same condition as before as regards hearing. To-day, made another series of observations of the animal while in its cage, and also after it was removed and placed in a small cage on table. Loud whistling, knocking, ringing electric bells in the neighbourhood failed to elicit any sign of attention. Occasionally, as before, there was an appearance of a start, or a look round, apparently conditioned by the noise, but repeated experimentation showed that this was pure coincidence."

I was not a little amused at listening to Professor Schäfer's comments on the above record which I read before the Neurological Society. First, he suggested that the notes themselves showed that the animal was not really deaf. And this, I admit, was in some respects an intelligible and fairly defensible position to assume. For doubts were constantly being raised, as the record freely states, in my own mind as well as in that of other observers.

It was only after long and repeated investigation, and variation of tests, that our doubts as to the animal's true deafness seemed fairly dispelled.

The only reaction which might be interpreted as a sign of some obscure sense of hearing was the occasional start coincidently with a loud or shrill sound made in its neighbourhood. But even granting that this was not mere coincidence, it might be due to some remnant of the centre, or co-exist with abolition of true auditory perception, and resemble the reaction to loud sounds which may be elicited in animals entirely deprived of their cerebral hemispheres, as Flourens and Longet demonstrated long ago.

But almost in the same breath Professor Schäfer next suggested that we had in truth lighted on a specimen of a monkey stone-deaf before the operation. It has been said that a dead donkey is difficult to find. A deaf monkey will be a greater curiosity. And when Professor Schäfer succeeds in finding one, it will not help him in his argument, as this animal's hearing powers were, as the record shows, well established before it was submitted to operation.

Professor Schäfer does not in his paper expressly repeat his brilliant inspiration, but, with a generosity which I do not fail to appreciate, he sums up his estimate of the evidence which Professor Yeo and I have given as to the localisation of the auditory centre, with the declaration of his belief, that he is "justified in asserting that the supposed localisation of the auditory perceptive faculties in the temporal lobe in monkeys, has no experimental evidence in its favour" (p. 377). Without disputing the accuracy of the views of Munk and Luciani, who have satisfied themselves that lesions of the temporal lobes in dogs cause total abolition, or profound

impairment, of the sense of hearing, he apparently takes comfort from the fact, that these physiologists have not actually experimented on monkeys. I am not concerned to speak for Munk or Luciani—believing, as I do, that they are both wrong as to the exact definition of the auditory centre in the temporal lobe—but they themselves, and doubtless all who believe in comparative anatomy and physiology, have no hesitation in extending their conclusions to monkeys and man. For if the localisation of an auditory centre is true of dogs, it must *à fortiori* be true of higher animals, in which cerebral localisation is more conspicuously manifest.

If, however, it should still appear that the results of experiments on the lower animals are not absolutely conclusive, let us turn to the records of human pathology, which is, after all, the *ultima ratio* in questions relating to subjective conditions, not clearly indicated by external manifestations. Deafness from cerebral disease is undoubtedly a rare occurrence. That this should ensue, requires the existence—rarely happening—of symmetrical lesions in both hemispheres; for unilateral lesions of the auditory centre do not appear to cause, at least permanent, affections of hearing. But as destruction of the motor centres of articulation in the left hemisphere causes aphasia, so destruction of the auditory centre on the left side causes the now universally recognised condition known as "word-deafness," a condition alone explicable on the hypothesis of the existence of an auditory centre. In every instance of this kind which has been examined post-mortem, the destructive lesions have involved the temporal lobe, and in all, the superior temporal convolution.

I have been able to find only two uncomplicated cases of symmetrical lesion of this region in both hemispheres, but both are of transcendent importance. Shaw[1] has recorded the case of a woman, ætat. 34, who, two months before her admission into his asylum, lost power in the right arm, and soon after had a sudden apoplectic seizure resulting in loss of speech and deafness. The loss of power in the right hand soon passed off. She became incoherent, more or less

[1] 'Archives of Medicine,' Feb. 1882.—Abstract in 'Brain,' Vol. V., 1882-83, p. 430.

maniacal, and demented. On admission, she was found to be perfectly *deaf and blind*. She died of pneumonia a year afterwards.

Post-mortem examination showed *complete atrophy of the angular gyri and superior temporo-sphenoidal convolutions of both hemispheres* (see figures accompanying the paper). The grey matter of the atrophied regions had entirely disappeared, leaving the outer layer attached to the pia mater, with a cavity underneath formed at the expense of the grey matter.

The cranial nerves were normal in appearance, the optic nerves showed increase of the connective tissue septa, atrophy of the nerve-fibres, and spaces filled with a colloid-like material.

This case may be objected to, as not being absolutely conclusive, owing to the fact, that possible disease of the organs of hearing is not expressly disproved in the record; though the sudden onset of deafness coincidently with the cerebral seizure would almost of itself negative such a hypothesis.

No such objection can be urged against the following case reported by Wernicke and Friedländer.[1] As this case is so important, I give the following particulars at some length.

A woman, aged 43, who had never suffered from deafness or affection of vision, was attacked on the 22nd of June, 1880, with right hemiplegia and aphasia. She remained in the hospital until August 4th, when she was discharged. At this time the patient could speak, but she spoke unintelligibly, and was sometimes believed to be intoxicated. She not only could not make herself understood, but she could not understand what was said to her. She was received into the hospital again on September 10th, with slight paresis of the left arm. The right hemiplegia had entirely disappeared. The patient was looked upon as insane. She was absolutely deaf, so that she could not be communicated with. She died of an attack of hæmatemesis on the 21st of October. An extensive lesion was found in each temporal lobe, invading the superior temporal convolution on both sides (see figures

[1] 'Fortschritte der Medicin,' "Ein Fall von Taubheit in Folge von doppelseitiger Laesion des Schläfelappens," Band i., No. 6, March 15th, 1883.

accompanying paper). The rest of the brain exhibited no abnormality; nor were there any conditions leading to increase of the intra-cranial pressure and possible affection of the cranial nerves. It was proved that the patient had previously enjoyed excellent hearing. Her total deafness occurred rapidly in connection with the other indications of cerebral disease. In order to exclude possible local affection of the ears, these organs were submitted to examination by Professor Lucas. The result of this examination was practically entirely negative, only a slight dry catarrh being found, and nothing locally to account for deafness.

This case will, I venture to believe, be held to justify the conclusions drawn by the authors themselves, viz. "that the auditory nerves ultimately end in the temporal lobes, and that double or bilateral lesion in these lobes causes complete deafness. It may therefore be stated with all certainty that the temporal lobes are the cerebral centres of hearing."

And I should myself go further and say, that the case is entirely in harmony with my own more strict localisation of the auditory centre in the superior convolution of this lobe.

The experimental evidence in favour of the localisation of special centres of smell and taste, which has, up to the present, been adduced by myself in the case of monkeys, and by Munk and Luciani in dogs, cannot be said to possess such precision in respect to the position and extent of these centres as not to stand in need of further investigation. My own work in this direction has been mainly that of the pioneer. But these experiments, with all their deficiencies and imperfections, taken by themselves alone, furnish at least a strong presumption, that the olfactory centre is situated in the hippocampal region of the temporal lobe, at its lower extremity. But when taken with the comparative development of the hippocampal or pyriform lobule in *osmatics* and *anosmatics*, and the anatomical relations of this region to the olfactory tract, and anterior commissure, they constitute a body of evidence in favour of the localisation of the olfactory centre mainly, if not exclusively in this situation, which I venture to think will not be considered as materially weakened by Professor Schäfer's inability to detect any impairment of the sense of smell in

those animals in which he professes to have completely removed this lobe.

Investigations as to the sense of smell in the lower animals are perhaps more difficult than that of any of the other sensory faculties, seeing that the external manifestations of olfactory perception proper—apart from those caused by substances possessing some degree of pungency and not strictly pure olfactory stimuli—are not so evident as to ensure detection without a long previous study and minute acquaintance with the habits of the animal.

But inasmuch as Professor Schäfer, while unable to confirm, is also unable to adduce any facts opposed to the positive results obtained by Munk, Luciani and myself, it does not seem necessary to discuss the question at greater length. I would only remark that, if Professor Schäfer is right in his conclusions that the *whole* of the temporal lobe, including the cornu Ammonis and hippocampal lobule, may be removed without abolishing or impairing the sense of smell, he must be considered as having proved more than he could wish:—viz. that comparative anatomy is a delusion and a snare.

The exact position of the gustatory centre is admittedly less clearly defined than any other.

I have related two experiments[1] in which the lower half of both temporal lobes was extensively disorganised. In these animals no reaction was induced by placing on the tongue such substances as aloes, citric or acetic acid, which in normal monkeys excite active expressions of disgust and discomfort. I do not profess to have determined the connection between these symptoms and the destruction of any particular region more precisely than that above described; nor were the animals kept sufficiently long alive to decide the question as to their permanency, but the observations are not without important significance.

Professor Schäfer objects, that the animals "were in an entirely abnormal condition when tested, nor was the testing performed with purely sapid substances, but with acid and aromatic particles."[2] The only abnormal conditions present

[1] Experiments 14–15, 'Phil. Trans.,' vol. 165, Part 2

[2] P. 378, footnote.

in these animals, different from others similarly tested, were the position of the lesions:—viz. in the lower half of the temporal lobes. And even if aloes, colocynth, citric and acetic acids are not "purely sapid substances," but "also acid and aromatic particles," the objection to their use seems somewhat irrelevant, seeing that no reaction whatever was induced by any of them.

Let us now turn to Professor Schäfer's utterances in respect to the visual centre of the cortex. The chief point at issue between him and myself is the anterior boundary of the visual zone. While he considers it as being sharply bounded by the parieto-occipital fissure, I maintain that it embraces also the angular gyrus—the two constituting together what I have termed the occipito-angular region.

With respect to the effects of electrical irritation of this region, it is true that while *invariably* obtaining movements of the eyes from the anterior and posterior limb of the angular gyrus—and in this I am absolutely confirmed in every instance by Luciani and Tamburini—I did not observe similar movements, or other external manifestations, on irritation of the occipital lobes alone. But inasmuch as Luciani and Tamburini did *occasionally* observe similar movements of the eyes on irritation of the occipital lobe, though less marked than in the case of the angular gyrus, and as Professor Schäfer seems to have had no difficulty in always obtaining the same, I am not disposed to question the accuracy of their results, and will admit that I may have overlooked them, though actually present in the ten animals on which I experimented; or that the parts may have lost their usual excitability owing to some temporary conditions. But this admission only strengthens the position which I have maintained. For if the same, or similar, movements are capable of being excited from the occipital lobe, as well as from the angular gyrus, the presumption is that the regions in question have a community of function. And I am glad to observe that Professor Schäfer adopts my view, that these movements are the expression of subjective visual sensation.[1]

Professor Schäfer is unable to confirm the results obtained

[1] Proceedings of the Roy. Soc., March 1, 1888.

by myself (and in conjunction with Professor Yeo) on destruction of the angular gyrus; and he is inclined to attribute such effects as we have observed to mere functional perturbation of the true visual centre situated in the occipital lobe.

I will, for the purposes of argument—but only so far—admit that the temporary total blindness consequent on bilateral destruction of the angular gyrus, may be explained in the manner suggested by Professor Schäfer; and I will therefore exclude these experiments from consideration. But inasmuch as it is held by Professor Schäfer, Munk, and others, that unilateral destruction of the visual centre always causes homonymous hemiopia, and not crossed blindness, or amblyopia, the results of destruction of the angular gyrus, if merely attributable to functional perturbation of the occipital lobe, should in all cases assume the form of hemiopia. How then are the following facts to be explained in accordance with this hypothesis?

In one animal[1] the left angular gyrus was cauterized with the galvanic cautery.

"The left eye was secured, and the animal allowed to recover from stupor. At the end of half an hour, it was evidently wide awake, but would not move unless touched. At this time it was removed from its cage and placed on the floor, whereupon it began to grope about in a sprawling manner, knocking its head against every obstacle. After some minutes of this behaviour, it subsided, and refused to move. It made no sign of fear at threatening gestures, and did not wink at a thrust of the finger at its eye, until the finger almost touched the conjunctiva, when the usual reflex closure occurred. Half an hour later, the same tests were employed with precisely the same indications of total loss of vision. At the end of still another half an hour, while it was lying quietly in its cage, it was gently laid hold of without noise to attract its attention, whereupon it bounded away with an expression of fear and surprise, and ran full tilt against the leg of the table, where it remained groping and sprawling for a few minutes. It then started off, and this time ran against the wall, against which it sprawled helplessly. Similar things are repeated. It gave no sign of perception when it was cautiously approached without noise. But when a slight noise was made with

[1] Experiment 5, 'Phil. Trans.,' Part 2, 1884.

the lips quite close to it, it darted off and came against the wall as before, where it lay down. Half an hour later, while it was resting quietly in a corner, with its eye open, the light of a lantern was flashed in its eye, but it gave no sign. Creeping up to it cautiously without exciting its attention, the observer made a slight whisper close to its face, whereupon it peered eagerly; but evidently remembering the results of running away, it crouched down and would not move. Half an hour later, when it was quiet in its cage, it started suddenly on being touched, and ran its head into a corner, where it crouched.

"Next day, its left eye being still closed, it showed unmistakably the possession of vision with the right eye. It laid hold of things as usual, and ran about the laboratory in every direction, passing obstacles right and left with perfect precision, and ducking its head to pass underneath bars as it ran along the top of the waterpipes of the laboratory. No defect of vision, amblyopic or hemiopic, could be detected."

In another animal,[1] the left angular gyrus was cauterised up to the parietal-occipital fissure (the posterior part of the corpus callosum being also divided at the same time).

"The left eye was securely closed, and the animal allowed to recover from its narcotic stupor. In half an hour it began to move about spontaneously, although rather unsteadily. An hour and a half after the operation it walked about the laboratory, knocking its head against legs of chairs, and other obstacles in its path. When a piece of apple was held under its nose, it grabbed it and ate. It continued to walk about here and there, every now and then coming to a dead halt full tilt against the wall. Three hours after the operation, it again, in running about the laboratory, came full tilt with its snout against the wall, where it rested. While it was resting quietly, we crept up to it; but the animal, though with eye wide open, and looking towards us, made no sign of perception. Threatening grimaces were likewise without effect; but on making a noise with our lips, the animal seemed alarmed, peered forwards, and yet, though it came quite close to our faces, seemed to see nothing. It was tried to right and left in the same way, but there was no sign of vision, to one side or the other. Next day, the left eye being still closed, the animal ran about in every direction, ducking under bars, passing objects to right and left with the utmost precision, and never once knocking against anything one side or the other. Not the slightest impair-

[1] Experiment 7, 'Phil. Trans.' part 2. 1884.

ment of vision could be detected; and it was able to pick up the minutest objects lying about its cage, or thrown down near it."

Similar results were obtained in many other instances, and if in some they were even more transient, this may have been due to less extensive destruction, or perhaps to differences in animals as to the relative importance of this region in the ordinary exercise of their visual faculties.

But these facts, carefully noted and faithfully recorded by Professor Yeo and myself, are altogether unintelligible on the hypothesis that they are merely due to functional perturbation of a centre, the destruction of which causes only hemiopia. If, nevertheless, it should still be asserted that the results, being so transient, indicate rather functional perturbation than the cross relationship which I hold to exist between the eye and the angular gyrus, it would be natural to expect, that the much more severe operation of actual extirpation of a large portion of both occipital lobes would produce a greater disturbance of the visual centres than the mere erosion of the surface of the angular gyrus. How then shall the following facts be explained?[1]

"The occipital lobes were exposed on both sides in a monkey, and the surface exposed destroyed by the cautery, which was also passed deeply into the interior of the lobes so as to cause as much disorganisation as possible. Care was taken not to injure the angular gyrus. The operation was completed at 3.30 P.M.

"4.10 P.M.—The animal after lying in a state of stupor until now, begins to move, but staggers a good deal. The eyes are open, and the pupils are dilated. It indicated consciousness by turning its head when called to.

"5.45 P.M.—Gives emphatic evidence of sight; ran away when I approached it, carefully avoiding obstacles. Seeing its cage door open, it entered, and mounted on its perch, carefully avoiding the cat which had taken up its quarters there. Tried to escape my hand when I offered to lay hold of it, but picked up a raisin which I had left on the perch."

Here is a case of extensive destruction of both occipital lobes, and yet in little more than *two hours* after this serious

[1] Experiment 22, 'Phil. Trans.,' vol. 165, Part 2.

operation, the animal gave the most emphatic evidences of precise vision. Or take the facts of the following case[1]:—

"The occipital lobes were exposed in a small, rather weakly monkey, and the lobes severed by a perpendicular section with hot wires about a quarter of an inch posterior to the parieto-occipital fissure, so as to avoid all interference with the angular gyrus. The operation was completed at 4.30 P.M., the animal having by the time the wound was dressed almost completely regained consciousness.

"4.45 P.M.—Began to move about in rather a staggering manner, but exhibiting no muscular paralysis.

"4.55.—Can see quite well, as it avoids obstacles, and when removed, regains its place by the fire."

Here is a case in which, after removal of at least two-thirds of both occipital lobes, the animal indicated its possession of vision *within half an hour* after the operation.

Or take a third case[2]:—

"In this animal the occipital region was exposed on both sides, and *both occipital lobes* were severed with the galvanic cautery and scooped out bodily. The line of incision in both cases passed between the anterior extremity of the first occipital, and the parieto-occipital sulcus. The portion removed on the right side weighed 1·9 grammes; that removed on the left weighed 2·1 grammes [fully two-thirds of the whole].

"In half an hour after the operation the animal sat up and wanted to move about, but it was kept still, wrapped in a blanket to prevent the risk of hemorrhage. Both eyes were open equally, the pupils smallish, and the conjunctival reflex equally distinct on both sides. For two hours, though it was looking about as though it saw, no definite test of vision was made; but at the end of that time, it went and sat down beside the next cage, and allowed the other monkeys to handle it and examine its head. It then, of its own accord, went into its own cage and sat down. A piece of apple was thrown into the cage, and though it fell a full arm's length away, the animal, without the slightest hesitation or want of precision, put forth its left hand, picked it up and ate. Half an hour later, similar tests were made. A piece of apple was thrown near it; this it took and began to eat: on the piece being taken

[1] Experiment 23, 'Phil. Trans.,' vol. 165, part 2.

[2] Experiment 9, 'Phil. Trans.,' 1884.

from it, it seized another piece lying in the cage and ate this, and having finished it, came forward a few steps and picked up another piece lying on the floor."

There was thus the most complete proof of retention of vision. Here again, *within two hours* after removal of at least two-thirds of the occipital lobes on both sides (see photograph plate 21, Fig. 15) the animal indicated sufficiently clear vision to be able to pick up a minute object lying on the floor. Within, therefore, the time at which animals with unilateral or bilateral destruction of the angular gyrus are blind on one or both sides, the much more formidable operation of destruction of the greater portion of both occipital lobes leaves the animal's power of vision practically unimpaired, so far as ordinary tests are concerned. It is surely therefore unreasonable to ascribe the effects of mere erosion of the angular gyrus to functional disturbance of the occipital lobe.

I do not, however, argue—as I at first believed—that the occipital lobes do not form part of the visual centres, but conclude from these facts, that greater relative disturbances of vision occur from lesions of the angular gyri than from much more extensive lesions of the occipital lobes. They show also, what an enormous amount of destruction can be inflicted on the sensory or perceptive centres of the cortex without obvious impairment of their functions.

I have always, in contending for the negative effects of extensive removal of the occipital lobes, specially made the proviso, that the lesions shall not extend up to the parieto-occipital fissure. For I was myself the first to point out that, when the lesions extended so far forwards, impairment of vision, or even total blindness, ensued—results which I attributed to direct implication of the angular gyrus, or its medullary connections.

That this is the real explanation of the total blindness obtained by Munk and Schäfer on removal of the occipital lobes, it will not, I think, be difficult to demonstrate, and that, too, even from Professor Schäfer's own facts alone.

If his Figure 3, p. 368, be examined, which represents the extent of the lesion in a monkey said to have been rendered

totally and persistently blind, it will be seen that the lesion extended on the occipito-temporal aspect beyond the anterior limit of a line drawn along the course of the parieto-occipital fissure. On both sides, and particularly on the left, the lesion extends well into the collateral fissure and beyond the anterior boundary of the occipital lobe proper. (A similar condition, even more marked, exists in the brain of the hemiopic monkey, Fig. 2, as I have found on personal examination.) If the next figure—Fig. 4—be examined, in which vision was not abolished, it will be seen that the lesion, though it extends slightly beyond the limits of the occipital lobe proper, does not extend so far forwards as in the first case.

On this the whole question turns.

Professor Schäfer admits that, with the exception of this difference, the removal of the occipital lobes was equally complete in both cases. It is obvious, however, that if it were merely a question of removal of the occipital lobes, the results should have been precisely the same. The conclusion to be drawn from this is that the total blindness was due to implication of the region lying in front of the line of the parieto-occipital fissure. Other facts, some of which have also been supplied by Professor Schäfer himself, place this beyond all doubt.

I have recorded several instances[1] in which in attempts to remove the hippocampal region, I have observed, in addition to other symptoms, hemiopia of greater or less degree and duration. In one of these[2] there was only superficial erosion of the surface of the inferior occipital convolution; but at the anterior extremity of this gyrus, where it becomes continuous with the middle temporal convolution, and just at the lower extremity of the parieto-occipital fissure, the lesion became more deep, and thence extended along the middle temporal convolution, detaching all below this.

In another case[3] the occipital lobe was altogether uninjured, the lesion commencing just below the inferior extremity of the parieto-occipital fissure.

[1] See Experiments 27, 28, Photographs, Fig. 125 and Fig. 134, 'Phil. Trans.,' part 2, 1884.

[2] Experiment 27, Figs. 125–132

[3] Experiment 28, Fig. 134, l. c.

Professor Schäfer tells us that he too has observed hemiopia of temporary duration as the result of extensive lesions of the temporal lobe.[1]

It is evident that the hemiopia observed in these experiments does not indicate the position of the visual centres. For from these facts alone we should have as much reason for placing the visual centres in the temporal as in the occipital lobe.

There can be no doubt that the hemiopia in these cases was due to the lesion invading the occipito-temporal region, where the inferior occipital becomes continuous with the middle temporal convolution.

This point is situated on a level with, and opposite, the entrance into the hemisphere of the optic radiations from the corpora geniculata and primary optic ganglia.

It is to section of the optic radiations in this region that Professor Schäfer's results are to be ascribed. It is these which were destroyed completely in the case in which complete blindness occurred, and only partially injured in the case in which a large amount of vision was retained. Professor Schäfer cannot say, that the degree of vision retained by the latter animal depended on the cortex of the occipito-temporal region not being completely removed. For he relates a case in which, after complete recovery from any temporary effects caused by extensive removal of the occipital lobes, the destruction of the angular gyrus—*without any lesion of the occipito-temporal region in question*—caused complete and persistent hemiopia (p. 363).

Professor Schäfer has himself furnished me with so many facts showing that the visual centres are not confined to the occipital lobes proper, but embrace also the angular gyri, that it seems unnecessary for me to insist further on the subject.

Indeed, he appears himself to have some suspicion that "fibres connected with the cortex of the adjoining parts of the brain (and especially of the angular gyrus) may curve backwards into the occipital lobe, and thus become cut off along with that lobe" (p. 372).

[1] "The nearer to the occipital lobe that any such lesion of the temporal lobe is carried, the greater probability is there of the occurrence of (temporary) hemiopia" (p. 372).

It is a pity that he did not examine his own facts a little more closely before committing himself as he has done. For he would have seen that the really effective lesions causing complete and permanent hemiopia—apart from conjoint destruction of the occipital lobe and angular gyrus—were those which severed the optic radiations in the position I have above indicated. And if Professor Schäfer will restrict his lesions to this region, without at all injuring the cortex of the angular gyrus or occipital lobe, he will obtain all the results which he attributes to removal of the occipital lobe itself.

While regretting for his own sake the attitude he has taken up on this question, I cannot feel otherwise than indebted to him for inducing me by his criticisms to re-examine, and, as the result, to re-affirm, with increased conviction, the views which I have previously enunciated.

Postscript.

The Editor of 'Brain' has received the following note from Prof. Schäfer, with reference to an objection raised by Prof. Ferrier to the term "Report" used by him in the January number of this Journal:—

"A statement in my Paper in the last number of 'Brain,' regarding a report by members of a Committee of the Neurological Society having been understood to imply that a Collective Report had been published, I wish to state that the report here alluded to was an individual expression of opinion supplied to me by those members of the Committee (Drs. Bastian, Brunton and Waller) who took part in the investigation, and which it was agreed to substitute for a Collective Report."

ON A CASE OF LOCOMOTOR ATAXIA FOLLOWED BY GENERAL PARALYSIS OF THE INSANE.

BY F. ST. JOHN BULLEN.

Pathologist and Assistant Medical Officer to the West Riding Asylum.

A. C., admitted August 4th, 1884, into the West Riding Asylum. Æt. 24.

Statements in the certificate:

1. Has delusions of pecuniary and other possessions; is excitable and restless.

2. Has been altered in his mind since a "Paralytic Stroke" three months ago, becoming irritable and irascible, slovenly in his habits. Would go out, half-dressed, if not prevented. Memory defective.

Family History.—His mother in Wadsley Asylum, suffering from chronic melancholia. His father a most intemperate and passionate man.

History.—Patient's occupation had been that of clerk to an estate agent. He had led a fast and intemperate life, but became a total abstainer four years ago, and remained so until about one month before his admission. About three months previous to this, he was seized with a fit of the following description:—"Whilst sitting at his office desk and performing some calculations, he experienced a sensation as of a blow on the left forehead, and fell off his seat unconscious, and remaining so for about five hours. Subsequent to the fit, his right limbs, especially the leg, were partially paralysed. The act of manual prehension was, on both sides, inco-ordinate, his caligraphy extremely bad, articulation stammering and lisping. No aphasia existed, but a marked effort was required for him to utter words. All parts of the body were numbed."

These symptoms passed off in about three weeks, but he had suffered a great change in his moral character. He would go out in the street half-dressed, use abusive and threatening language to his wife, and, for several weeks prior to his admission, indulged

in reckless debauchery. He complained after the fit of nearly continuous and severe cranial pain.

State on admission.—Bright, cheerful, conversable, and apparently of good intellectual powers. Explained the delusions mentioned in the certificate as "chaff," which he had uttered in response to some banter on the part of the workhouse porter. He denied any pecuniary or other possessions, but gave an exalted account of his mental abilities, saying he considered himself the equal of any living classical scholar, that he was Senior Classic at Cambridge in 1878, could speak seven languages and many dialects, and was a first-rate arithmetician. He manifested no failure of memory.

As regards physique, he was of medium height, rather poorly nourished; hair and irides dark brown; complexion sallow. Features clear cut, and expression intelligent. Both pupils dilated; the left nearly twice the size of the right, and the more active to light. Both reacted well to accommodation; sensory irritation produced no dilatation. His gait was somewhat staggering All the deep reflexes absent; superficial present. No want of tone in facial muscles, no defect of articulation. Respiratory, circulatory and genito-urinary systems were normal.

Progress of Case.—For the day after his admission he was quiet and orderly, but subsequently he became restless, interfering, abusive, unreasonable and excited; complaining after of much cranial pain. His statements as regards his powers as a linguist and classical scholar, etc., were found to be utterly untrue, and he admitted himself that his statements were "bounce." He was very untidy in his dress, constantly endeavouring to escape; had attacks of excitement, during which he would break windows, etc. After about three weeks of this behaviour, he became more reasonable and composed; no longer destructive; admitted all his previous boasting; made no more efforts to escape, and slept better. The left pupil continued the larger; in both, consensual, light, accommodatory and ocular movements were fairly performed. The tongue, protruded straight, was tremulous; speech shaky with an occasional trip in articulation. The gait markedly ataxic, unsteady, swaying to and fro; the feet lifted and set down jerkily and forcibly. Stood with eyes closed and feet approximated, he tottered and would have fallen if unsupported. No anæsthesia nor analgesia. He complained of difficulty in micturition at times; none in defæcation; also of frequent nocturnal seminal emissions. He stated that he had experienced diplopia both before and since admission, but more since.

After a lapse of three more weeks he became again excitable and emotional, declaring he was perfectly well; trying to escape

through a window; was threatening. Complained still of the frontal cephalalgia. Soon after this, he spontaneously described severe lancinating pains darting through both knees, and mentioned anomalous sensations in his tongue and mouth, for which there were no objective signs to account.

The left pupil was now acting less well to accommodation than the right.

His importunate behaviour influenced his wife to take the responsibility of his home care, and he was discharged on November 13th, 1884.

He was readmitted on March 19th, 1886. Had been in the workhouse for some time, and in the certificate was mentioned as troublesome, quarrelsome, and complaining. On admission, the left pupil was still the larger one; both reacted to accommodation and consensually; the right, sluggishly to light; the left, not at all. The tongue was tremulous and the facial muscles also when he became at all emotional. Deep reflexes absent; the superficial blunted. Cutaneous sensibility apparently unimpaired from objective trials, but he complained of some numbness in the left foot. The calf muscles were atonic; gait markedly ataxic; progression performed on a wide base; heavily descending heels, and constant glancing at the ground and clutching at neighbouring objects for support. The lumbar curve was increased, a plumb-line from between the scapulæ falling one-and-a-half inches behind the buttocks. His handwriting was good, but a little jerky. Was unable to touch his nose with the index finger when his eyes were closed, or to bring the digits of the opposite hands approximately together.

Fairly quiet and free from troublesome propensities for about ten days after his admission, at the end of this time he again resumed his old querulous, complaining behaviour, making constant accusations against every one brought into contact with him. The lightning pains in his lower extremities were now frequent, and caused him disturbed nights; the seminal emissions were rarely present. About this time obscure dyspeptic symptoms arose. The pupillary movements were very incoordinate, especially on any attempts at convergence; ocular movements unaffected. He gave accounts of a momentary giving way of the right leg, accompanied with dimness of vision and confusion which occasionally befel him, and of his tongue being drawn to the right side of his mouth irresistibly at times, this accompanied with a sensation of its being too large for his mouth, feelings of chilliness and sickness and anomalous feelings about the face. Three weeks subsequently to the date of these last notes the lightning pains

which extended down the thighs to the soles of the feet were accompanied with indubitable gastric crises. Occasional twitchings about his hands were noted, but these he stated had existed for nearly nine months. The succeeding month both lightning pains and gastric crises forsook him nearly entirely, but his complaining and accusing were constant; he developed delusions of suspicion and persecution on the part of the attendants, made entirely unprovoked attacks on helpless old patients, and at times had paroxysmal fits of passion, and indulged in foul and abusive language. Refused his food occasionally, but only for one or two successive meals at a time. Always intensely plausible and self-excusant.

Five months later the fulgurant pains returned, and now the upper extremities were involved; he described his legs as being shot out during the paroxysm, and complained of sharp, local pain over the area of distribution of Wrisberg's nerve at the elbow and of hyperæsthesia there. No superficial tenderness was present along the course of the nerve trunks, nor vaso-motor signs. He had occasional difficulty in emptying his bladder; the gastric crises had not returned at this time, but after two months' lapse they again set in; he could not sleep, even with the aid of chloral, and morphia which he had formerly taken to subdue the pain, he now rejected, persistently declaring that the medical officers were in league to poison him. He was excessively emotional, voice always very tremulous; his sight he described as not allowing him to read for more than five minutes at a time; the ataxia had so progressed as to render locomotion almost impossible.

The case now rapidly progressed to a conclusion. Rectal crises were superadded to the gastric, and these gave rise to fresh delusions on his part; he refused his food partially, and then, later, entirely. Fed once by the stomach-tube such distressing laryngeal symptoms were occasioned that its use was precluded for the future, and nutrient enemata were resorted to. He never spoke again, and remained in a pallid, cold, almost collapsed condition, with very feeble and irregular pulse. Required catheterisation often, and finally entirely. Ten days previous to his death, slight twitchings of the right side of the face set in, followed in twenty-four hours by severe general convulsions starting on the right side. These lasted about two minutes, and facial twitchings persisted for some time afterwards, but ceased under the influence of chloral nearly entirely. The next day he was able to swallow some liquid food; in the evening, however, the twitching again occurred, followed by a general convulsive seizure. During the next two days he had sixteen convulsive

seizures; the day after, they were diminished by the use of chloral to four, the next, to one. He seemed in pain, moaning, and drawing up his legs in bed; constantly contorting his features. On the left side the mouth is spasmodically retracted, and there is considerable ptosis of the left upper lid; the pupil on this same side, as throughout, the larger. He now took a little liquid food by the mouth. Little change but for the worse now occurred; partly silent, partly moaning during the remainder of his life, he uttered from time to time a sharp piercing cry, which probably indicated a return of lightning pains; was always hostile. The spasmodic twitchings of the face continued; signs of vaso-motor paralysis on the right side were present; the temperature in the axilla 100·5, as compared with 100·2 on the left. Bed sores over the external ankle and sacrum rapidly formed, and death ensued.

Autopsy.[1]

Spinal Cord.—The membranes were slightly thickened in the lower portions of the cord, and more noticeably so in the cervical region. Here, too, the vessels were obviously enlarged and engorged with blood, and some, though not by any means marked, loss of transparency had taken place. The pia in the upper cervical segments was pretty firmly adherent.

The cord is small, firm and of irregular contour in the upper and middle dorsal portions. The lumbar segment is, in comparison to the cervical, much shrunken, indicating the further advanced condition in this situation of the lesion.

The posterior columns show disease throughout their entire length, of the nature of grey degeneration, quite plainly visible to the naked eye. Sections were taken at short intervals along the cord, both transverse and vertical, and were stained with aniline blue-black; Weigert's process was also used. The former method of staining gave as usual very perfect results, and rendered accurately every detail of the pathological change. The postero-median and postero-lateral columns are seen to be deeply sclerosed, the former in their whole vertical extent, the latter only partially, in their uppermost portions. The lesion also affects other portions of the cord to a less extent.

In the region of irregular contour, viz., the dorsal, the

[1] See plate and letterpress at the end of this article.

anterior cornu of one side has suffered atrophic change and is reduced in width by at least, one-third. Its corresponding posterior cornu has likewise shrunken.

An immediately noticeable feature is the greatly increased vascularity existing throughout the cord-length in the posterior columns and grey matter. Nor is it absent from the lateral tracts. A description of the entire vessel changes will be given later. In the lower portions of the lumbar segment, the posterior median fissure is open through its dorsal half; in the rest of the cord it is closed.

Lumbar region—Lower portion—Posterior columns.—The whole of these are deeply stained by the aniline dye, with the exception of the commissural zones, less affected, a tract of nearly healthy tissue lying between the posterior cornu and the diseased parts of the postero-external columns, and lastly a small oval area occupying the columns of Goll at the edge of the median fissure towards its peripheral extremity. This zone appeared quite healthy. The depth of staining in the diseased localities is due to connective tissue increase, both fascicular and corpuscular, to a quantity of amyloid bodies, to absorption of the dye by the myeline of the nerve fibres, and to a less extent to the increased number of hypertrophied vessels. Every degree of involvement of the nerve-tubule is seen from the healthy specimen to that in which the axis-cylinder is the sole remnant. In the peripheral parts of the columns, the normal structural characters are replaced by dense fascicular fibrous tissue, and here are crowded corpora amylacea. Immediately subjacent to this are scattered large medullated fibres, whose myeline has remained unstained. The great majority of the parenchymatous structure is extremely atrophied and distorted in shape by the shrinking of the interstitial tissue; often nothing remains but axis-cylinders darkly dotting the fasciculi of the latter. In the commissural ends of Goll's columns, the connective tissue is somewhat increased in the form of numerous islets from which branches radiate amidst mostly healthy fibres. The anterior portions of the internal radicular fibres could be traced with moderate ease; they showed an extreme varicosity in this part of their course. Much of the latter cannot be traced.

Sections of the posterior roots in their vertical axis show a great increase of the connective framework to have almost cancelled the nerve tubules. Where these are seen, they appear largely swollen in some instances, wasted in others; the axis not always visible.

Lateral columns.—There is a tract of sclerosis of triangular shape, one of whose sides is in apposition to the posterior cornu and whose base is at the periphery of the cord. From its basal portion is continued a marginal zone of degeneration which fades gradually and disappears at the anterior root zone. The place of the normal tissue is largely taken by close fibrous tissue throughout the above mentioned localities, and the septa, extending into the lateral column, are coarse and followed by numerous amyloid bodies, whilst young connective tissue cells are in abundance throughout the crossed pyramidal tract. There is not however here any obvious atrophy of the parenchyma.

Anterior Root Zones and *Cornua* appear free from disease.

Middle and Upper Lumbar Regions.—A progressive invasion by sclerosis of the posterior commissural zones is indicated by the increase of connective tissue and of capillary orifices. In the middle segment the small oval zone in the median line shows commencing involvement by the degeneration. A small artery surrounded by thickened pia passes on each side into it and delicate fasciculi of this connective tissue proceed between the nerve tubules. Appearing between the latter, and displacing them, are branching cells whose processes spread and anastomose in every direction. At this stage no primary affection of the nerve fibres was in any way apparent.

In the upper lumbar segment, it is seen that this zone is now completely involved in the prevailing lesion, but extending backwards from it along the edge of the fissure is a narrow bordering of fairly healthy fibres. The commissural ends of Goll's columns manifest a marked increase of both connective tissue and capillaries, the latter usually forming the centre of an islet of the former. A large number of nerve-fibres have undergone change and the neuroglia is often finely dotted with the axis-cylinders. The corresponding part of Burdach's column is less involved, but there are a number of diseased

fibres scattered about the field, independently at this level, of increase of neuroglia.

The marginal degeneration of the lateral columns gradually lessens from the lower segment of the lumbar cord upwards and in the sections cut from the lowest dorsal region is not visible. There is a progressive increase of interstitial tissue in the crossed pyramidal tract.

Lower Dorsal Region.—Posterior columns. The degeneration is still very intense, and as in the lumbar region involves in the greatest degree the periphery of the cord. The vascular changes are more evident than as yet seen, and capillary orifices are in great number, and the postero-internal and external arteries are enlarged and tortuous. The median commissure is freely channelled by large thick walled vessels.

The amount of neuroglia in the posterior commissural zones is somewhat decreased, compared to that in the last segment of the cord described, but is thrown into greater prominence by the larger number of healthy fibres present.

Anterior columns and grey matter. These showed changes of the same nature as, but to a less extent than, those presently to be described.

Upper Dorsal Region.—Posterior columns. As compared with the rest of the zones at this level and its corresponding area in the lower dorsal segment, the tract including the periphery of the columns, the posterior nerve roots and the part of cord just internal to them, is less degenerate, and the lesion is tending to centre itself in the columns of Goll. In the commissural zones the number of healthy fibres is again reduced. The connective tissue is comparatively and relatively in excess over that in the upper lumbar region, and forms the predominating element in the field. The result of its contraction on the nerve-tubules is seen in two forms; atrophy as elsewhere, and a large swelling of the fibre which becomes indistinctly defined, of a dull stained appearance, and its axis swollen, wanting clearness, often hardly differentiable. These bulgings of the tubule are usually surrounded by an especially dense zone of connective. These appearances of degeneration extend into Burdach's columns, but are less in degree and area. Here and there are isolated

tubules, or small groups of tubules, affected seemingly independently of neuroglia increase, but it is probable that these have been strangulated at a lower level.

Lateral columns. These presented in the region of the crossed tract the same indications of lesion as before detailed.

Anterior columns and grey matter.—The anterior and posterior cornua on the left side have undergone marked alterations, and the corresponding hemisphere is changed in contour, much flattened, and its transverse diameter considerably diminished. The left anterior horn is somewhat longer than its fellow, lessened in width by fully one-third, its summit club-shaped instead of square. The extensions from its outer side are almost absent; from the inner, very sparse. Hardly one healthy ganglion cell is visible; such as are seen are in all stages of atrophy, shrivelled, apolar and situated in vacuolar spaces, not seldom the latter are the sole indications of their former presence. The border of the posterior horn and intermediate tract is with difficulty defined, the grey matter merging into the white. The cinereal substance as a whole is friable-looking and broken up; so loosely combined is its texture that under a low-power it has a peculiar patchy-white unstained appearance. Both cornua are freely sieved by numerous ascending capillary vessels, whilst the substance of the anterior is much channelled by its enlarged arteries. The cells of the posterior cornu are also constantly shrunken and apolar, no trace of branching connections can be seen. In consequence of the atrophy of the outer part of the anterior, and the indistinct definition of the corresponding portion of the posterior horn, the two appear to lie nearly in a straight line. Clarke's vesicular column on this side had a friable and degenerated appearance, being involved in the lesion of the grey matter, and its cells are shrivelled, often extensively pigmented and many wanting. In a number of sections taken, the average drawn was left to right as twenty-nine to forty. The invasion of the groups occurring from the outer side; it is here that the remains of destroyed cells are mostly seen.

The posterior horn on the right side is far from the normal, its appearance simulated less markedly those on the left. The right anterior horn is deeply stained, but the contour is not

obviously incorrect. The cell groups in this region are normally most indistinct, and the number of cells sparse, but comparison with a healthy specimen favoured both a decrease and deterioration of them here.

The column of Turck on the left side was much smaller than that on the right, its comparative diminution being in about the same ratio as that of the corresponding cornu. There however, beyond this, was no obvious disease of its elements, parenchymatous or interstitial. Such condition was present from the lowest dorsal segment upwards, and in the decussation region is still to be traced.

Cervical Cord.—Four segments of this were examined.

Posterior columns. In the first and lowest the clearing up of the degeneration in the periphero-external part of Burdach's column is progressing. Goll's columns are densely sclerosed still, and at their commissural ends the increase of connective tissue appears greater than any yet seen, and extends right up to the commissure. This portion of both columns is sieved by the numerous capillary orifices.

The lesion in the two higher segments presented characters generally similar to each other, and those of one, the upper, are alone detailed.

The postero-internal half of Burdach's columns is nearly normal, a few atrophied marginal fibres, an increased vascularity and numerous corpora amylacea only constituting morbid signs. The internal half of its periphery shows many fibres, enormously swollen, whose axes are feebly stained, greatly hypertrophied, often lying at the extreme side of the tubule, even not visible. These are scattered amongst nerve fibres some of which are extremely atrophied, others compressed, mis-shapen, stained, and ill-defined. In the commissural parts of these zones a narrow strip of comparatively unaffected tissue separates the grey matter from the advanced degenerative changes. There is, however, even here, some excess of neuroglia, and a few large vessels are seen bounded by healthy fibres.

The degeneration in Goll's column extends up to the commissure; its characters differ somewhat in parts, as now described.

In the posterior third, roughly speaking, the changes

presented are those of universally wasted and degenerated nerve fibres, irregular in contour from compression by the fine fasciculi of connective tissue which are closely specked with the axes of the degenerate smaller tubules. In about the middle third there are many medium and small-sized fibres of fairly healthy appearance, whilst areas of intense lesional alteration of larger and smaller extent exist.

The degeneration in the commissural portion is very advanced, and the changes shewn are of a different kind. A darkly-stained ground of utterly degenerate fibres and excess of neuroglia is mottled with bluish-white patches or streaks formed by partially diseased tubules the myeline of which is still preserved though stained a little. The nature of these streaks is only discoverable on careful focussing, the refraction being altered. Scattered about are large pale, oval, or round discs; probably fibres undergoing colloid change; the axis can but seldom be brought into view. Lastly, there are a few tubules of approximately healthy appearance.

Vessels in number traversed the field both vertically and horizontally; those at the marginal parts of the diseased areæ are as, before, the centre of an islet of connective tissue.

The sclerotic shrinking of the posterior columns has torn open in many places the septum closing the median fissure, and this is also the case with the division between the postero-median and external columns. In these situations the arteries are much hypertrophied and their lymphatic sheaths crowded with cell contents, and the processes of pia into the cord are infiltrated with leucocytes.

Upper Cervical region.—Lateral columns. The crossed pyramidal tract is stained in excess of the usual deeper colouring of this region, owing to an increase in the amount of connective tissue and blood vessels. There is a tract of somewhat densely fibrillated tissue lying in front of the posterior cornu, which, passing outwards, extends along the periphery of the cord, narrowing gradually; opposite the level of the tip of anterior cornu it disappears. From it coarse septa extend into the column, numerous amyloid bodies accompanying them. Large deeply-stained branching cells extend their processes between the cross-sections of the tubules in the pyramidal tract, and

contiguous to them are fibres showing commencing change. The most noticeable feature in the field is presented by the vessels. They are scattered in considerable number throughout the whole lateral column, excepting its marginal portions, but are especially congregated in front of the posterior horn, and laterally to the anterior horn. Here the substance of the cord is very freely channelled. The appearances in the two lateral columns are not quite symmetric, the left not showing as much connective tissue increase, though no appreciable difference in the number of vessels. Flechsig's limiting zone is quite intact.

Anterior cornua and columns.—In the lower two segments of the cervical cord, pronounced changes are seen in the grey matter. The anterior horn of the left side is narrower than the right, and instead of a bold convex outer contour, is concave from the falling in consequent on the disappearance of the antero-lateral group. All the cell groupings in these segments were very indistinctly marked, and the cells for the most part shrunken, black, and with processes stunted, broken off or absent. The internal on both sides is less degenerated than the others; the antero-lateral is more affected, whilst the postero-lateral group is almost completely wanting. The cells in the central area are hardly distinguishable. These atrophic changes were nearly symmetric, but the right horn was a little the less involved. Many coarse vessels traversed the cornua; the cell groups are often, quite indifferently, broken up by some large branch, and it is observable that the higher up in the cervical cord sections are taken, the more does the vascular increase affect the grey matter.

Moreover another feature to be noticed is, that although there is throughout the greater vertical entirety of the lateral columns a marked excess in the vessel numbers, together with hypertrophy of their coats, yet nowhere is there any sign of accumulation in the perivascular sheath of lymph-cells, or such changes in general as are met with in connection with the vessels in the posterior columns. The central canal and the grey substance immediately surrrounding it presents a heaping-up of dark-stained endothelial cells, the canal being in much of its extent obliterated by them—this appearance applying to it in the entire length of the cord.

In longitudinally-cut sections of the posterior columns the medullated nerve-tubules are, more markedly at the periphery of the cord, of an extremely varicose appearance. This not seldom seems due to constriction by embracing fibrils of connective tissue, but more often independently of such a cause. The axis-cylinder is, at times, swollen and fusiform for a small part of its length, very tortuous, and like the nerve-tubule presents marked varicosities and constrictions.

ADDENDUM.—It was not found possible, at the date when the description of the lumbar cord was written, to procure sections from the extreme lower end in that region, owing to the friability of the tissue. By the use of a modification in the imbedding process, later on, satisfactory specimens were obtained. The sclerosis here is seen to be less diffuse, and to admit of localisation better. The internal radicular fibres appear to constitute especial lines of intensity; elsewhere it is perceptibly gathered around the vessels in their course, and these latter form a far more important feature in the field than they should in a healthy cord, in this region. The bordering of healthy fibres along the posterior median fissure extends further towards the commissure than in the sections of the lumbar cord described at the commencement of this paper.

Vascular system.—Its condition has been briefly alluded to as the various cord-segments have been described, but some additional notice may be given to this. It may be said, broadly, that scarcely a healthy vessel can be seen throughout either cord or medulla; this statement however involving the acceptance of hypertrophy and dilatation as evidences of departure from the normal. Such conditions as the latter are present, or perhaps frequent, in portions of the cord where no obvious disease of its structure exists, and, beyond having a certain bearing on the question of a general vascular lesion, do not call here for more remark. It is in the posterior columns and the grey matter that a change of interest has occurred.

In the commissural zones and grey matter this increase takes the form of very numerous pin-hole apertures of circular, defined, or punched-out appearance, the transverse sections of capillaries. These are in greatest number where the sclerosis is younger, and seemed in many parts the evident precursors of

the degenerative change. In the remaining area of the posterior columns the vessel orifices are less numerous but larger, and the majority of the arteries are met with running inwards in the plane of the section. The most prominent of these in the lower segments of the cord are the postero-internal and external arteries, which are greatly hypertrophied and enlarged, cut in more or less of their extent; that part of the former which lies along the posterior cornu is even more enlarged than any other, and is so in the lowest sections taken. The smaller vessels in this region have their walls collapsed and lumen obliterated by the shrinking of the sclerous tissue; in the higher parts of the cord the increase in number of the arteries is more readily seen. The median fissure is occupied by a large branch and an also large offshoot from this runs into Goll's column on each side.

In the grey matter of the posterior horns, capillary orifices are extremely numerous; that of the anterior horns is traversed frequently by large channels; seldom, nearly bisected by one. The commissures show groups of vessel mouths, and are almost interrupted by channelling in many sections. Occurring also with tolerable frequency are large circular excavations, probably the result of vessel changes.

In sections cut in the longitudinal axis of the cord, the posterior columns appear almost riddled with apertures, vacant or containing the remains of vessels. These openings are for the most part much in excess of the diameters of the latter, but their contents in these sections are mostly absent; around the margins, of dense sclerosed tissue, are numerous connective tissue nuclei and amyloid bodies. In the parts of the section corresponding to the commissural zones, the arteries seen in length are occasionally of a remarkably varicose outline, suggesting the affection described by Obersteiner as occurring in paralytics.[1]

Synopsising the vascular changes found throughout the medullo-spinal axis, the arteries are remarkably often tortuous; their coats much hypertrophied, the lumen sometimes dilated; sometimes invaded, even to obliteration, by conjoined thickening of the walls and contraction of the surrounding

[1] 'Rosenkranzform des Muscularisrohres.'

tissue. This thickening of the tunics is not uniform, being generally increased at the points of curvature, probably both from approximation of naturally more distant points, and from actual increase in the amount of tissue. The cut edge of the vessel has a homogeneous, ground-glass-like appearance as a rule.

In parts the adventitial coat has been drawn out, and is seen to be of a finely fibrous texture and thickly sprinkled with branching nucleated cells on its inner surface. The endothelium is pretty generally swollen and indistinct, and its cells detached often in mass into the lumen of the vessel.

Along the course of some of the smaller arterioles are seen young branching connective tissue corpuscles. These not only lay outside, but inside the perivascular sheath; in the latter situation in company with embryonic cells, their processes passing outwards between the nerve fibres. Throughout the cut edge of the vessel coat, cells with or without off-shoots are seen lying, occasionally protruding in part from the outer surface of the adventitia, or passing through the endothelial lining. In the last, are imbedded leucocytes at intervals.

The connective increase would suggest an origin in a proliferation of the cells of the adventitia, and in migration of lymph-cells through the vessel coats.

In the dorsal portions of the cord above them, these changes in the perivascular sheath are replaced by others of a marked nature. It is distended by large, pale, granular, unstained bodies, round or ovoid shaped; the nucleus in which is of almost equal size to that of the cell itself. With these in the space are embryonic cells, pigment and granular masses.

In the cervical segments, the accumulation is remarkable, and not limited to the perivascular space, but existing between the arterial tunics and splitting them up for a considerable distance, or else heaped up in limited areæ causing a local occlusion of the lumen. These bodies are apparently, in some instances, diffused into the surrounding tissue.

Throughout the medulla, pons, and crus, are met with, here and there, vessels whose outer border has a strikingly beaded appearance and double contour. Under a high power, such

characters are due to a single row of fairly regularly-sized small round bodies in the perivascular lymph-space.

The other vessels in these regions show frequently cells of less regular shape in the same situation, and less in number, or merely thickening of their coats, and cell proliferation.

Medulla.—The upward progress of the degeneration along the cuneate and slender columns is plainly visible, and the cells of their nuclei evidently atrophied and otherwise diseased.

The fibres constituting the zonular layer show throughout their extent a varicose outline, such as is seen in those of the posterior columns. Thickly scattered along their course are amyloid bodies and connective tissue nuclei, the distribution of these being continuous from the posterior columns into the nuclei of the pyramids and these latter indicating changes in excess of the allowable variations in healthy tissues. From these nuclear formations connective tissue nuclei follow the course of the fasciculi passing into the median raphe; there are they lost in the deep-staining of the latter, and no disease is traceable along its extent, but those fibres from its posterior part which penetrate the hypoglossal nuclei manifest tumefaction and varicosity to an extreme degree.

Those inner bands of the arcuate system, which, skirting the central grey column are to be traced into the pyramids, appear altered from the normal. They are narrower and their medullary sheath wasted, the fibrillæ having a thin, thready look; their outline is blurred and indistinct, by reason of the absorption to no small extent of the aniline dye.

In the lower regions of decussation, the sclerosis extends from the posterior columns around the caput cornu, investing it; higher up its position is occupied by the ascending roots of the fifth, and these latter, whose sectional area always presents a dark appearance from the scanty white sheath possessed by the tubules, are traversed by a considerable excess of fibrous tissue. The same increase of neuroglia and a complete, but not uniform, obscuration of the nerve-fibres is seen in the funiculus solitarius on both sides, and the fibres of those nerves which curve round this, their column of origin, are at this point remarkably varicose and swollen.

The central grey substance, in the region of the inferior

pyramidal crossing is much channelled by vessels. Here also a large transversely running fissure is placed just posterior to the central canal, completely bisecting the grey matter, and probably resulting from the friability of the latter produced by degeneration. Not seldom large cyst-like cavities appear; their nature in some is indicated by the adherence to their walls of portions of thickened vessel tunics.

The group of cells occupying that portion of the central grey substance placed postero-laterally to the canal is on one side degenerated; very few of the cells remain; they that do are shrunken or inflated, mostly apolar.

The ganglion cells of the anterior cornua and those scattered throughout the reticular formation, seem healthy, also those of the restiform body, hypoglossal, abducent, and facial nuclei.

The vagal nucleus of one side is degenerated; the tissue in which it is situated has the same whitish, friable look as that of the cornu in the dorsal region. Through several sections its cells are seen to be in different stages of decay, and compared with those of the opposite nucleus to be greatly diminished in number, the number also of those always pigmented appears decidedly increased.

In some successive sections is a large tortuous vessel, occupying the centre of the cell-group and breaking it up.

The affection of the corresponding vagal nerve is by no means so marked as that of its nucleus, but by the observation of a number of sections morbid changes in it became certain.

The area of its fasciculi is diminished, and the latter are often deeply stained; those fibres which join the main trunk from the substantia gelatinosa are especially changed, and their medulla appears almost completely wasted. The moniliform character of the diseased nerve tubules is very noticeable. At the border of the medulla, where the trunk crosses the zonular layer, its fasciculi are invaded by the connective tissue corpuscles and amyloid bodies which stud the latter as far inwards as the gelatinous substance.

In sections taken through the upper planes of the medulla the acoustic nerve and some part of the dispersed nuclear origin is diseased. Unfortunately one side of the section had

lost the nerve root; it was impossible to identify which. The sclerosis in the acoustic trunk is extreme; its area is composed of thick wavy bundles of connective tissue, the inter-spaces of which are occupied by almost unrecognisable remains of nerve-fibres imbedded in the coarsest meshwork of neuroglia; in the sèpta are running numerous dilated vessels. Those of its root-fibres passing between the gelatinous substance and the inner peduncular body are deeply sclerosed, those peripherally situated are less affected and signs of disease in them fade fast towards the nucleus. In several sections a long and coarse artery traverses the nerve-root for some distance. A strip of pia, adherent in all specimens, between the nerve and the body of the medulla, is seen to be considerably thickened, its vessels large and engorged with blood, and a number of leucocytes to be scattered in its meshes. The cell group of the posterior median acoustic nucleus lying to the inner side of the internal peduncular tract, it was easy to assure oneself in ten sections was degenerated, one being invariably correct in identifying the diseased area as to its side without the aid of the corresponding nerve. The change has mostly affected the large multipolar cells, which are shrunken and their processes stunted or absent.

The uppermost portion of the internal auditory nucleus, a small clump of cells lying beneath the grey floor, appears on the one side free from lesion, on the other it was entirely absent.

There is an increase of connective tissue in the pyramidal columns of the medulla in its lower planes, but it is gradually lost in the upper, and no lesions, except those described, are apparent.

Quadrigeminal region.—The tissue in this region is rendered so friable by disease that satisfactory sections are difficult to obtain. The peripheral part of the specimen on the left side has been injured a little during the course of preparation, no important structures are however damaged, and the respective sides of the mounted sections are made by it capable of certain identification.

The vascular increase here as elsewhere is very marked. In the mid-line behind the aqueduct is a large thick-walled

vessel, which may be said to represent the artery of the posterior median fissure. In the sections in which this vessel is not present, its position is occupied by a deep-stained band of fibrous appearance, on and around which are thickly-dotted amyloid bodies. These latter are very numerous throughout all the layers posterior to the aqueduct.

Although the entire medullary matter is over-vascular, yet this latter condition is more especially marked in its left half. Here a complete riddling of the tissues by thickened vessels has occurred.

In the anterior half of the left side of the chain of fibres constituting the descending roots of the fifth nerve, the fibres are much swollen, but clearly defined and unstained; the axis cylinder, however, cannot be detected in one of them.

The oculo-motor nuclei at the level of the nerve roots show considerable changes. The cells which are scattered amongst the fibres of the raphe are very few and deeply pigmented; the smaller elements which lay most posteriorly are still more obviously diseased, whilst the median and largest group is asymmetrically affected. On the right side its cells are thinned in number and atrophied, on the left the group is split up by a large coarse vessel which runs the whole anterior-posterior extent, and on each side of which the nervous structure is plainly diseased, but far more on the inner side, where the nerve-cells are almost absent.

The disease in this nucleus, although tolerably uniformly distributed, is intensified around the vessels, and the latter suggest themselves as an evident origin of the lesion. Both posterior divisions and the left median of it are especially invaded.

The dispersed cell formation, existing in the grey matter anterior to the aqueduct and below the level of the roots of the third, is prominently degenerated. Almost all the cells are extremely pigmented in a large number represented only in granular masses.

The fibres of the third nerve do not appear consistently diseased, but in some sections are fairly healthy-looking; in others the bundles are very meagre and threadlike.

Owing to the condition of the grey matter of the quad-

rigeminal bodies, the characters of its cells are rendered somewhat uncertain. The larger form, whose processes lying radially are understood to assist in the completion of the reflex arc between optic and third nerves, where distinctly seen do not appear diseased.

The Optic Nerve.—The sheath of this nerve is considerably thickened and the septa of neuroglia very much so. The latter appear as coarse, wavy, connective tissue, freely permeated by vessels, infiltrated in the vicinity of the latter by leucocytes and branching into the nerve bundles in all directions. These last are also invaded by arteries independently of the septa, and are plentifully dotted with an increased number of somewhat enlarged connective nuclei.

The external layer of some of these arteries has undergone so much thickening and its laminæ have become so wavy and differentiated as to resemble very closely the processes of connective tissue. The perivascular sheath almost invariably allows its contents to be recognised, in the form of swollen, largely nucleated, endothelial cells and lymph corpuscles. The vessels themselves are mostly engorged with blood or filled with disintegrated endothelium. In some cases their walls show the change noted elsewhere, in the description of the vascular system, viz. the beaded contour. This, as before said, depends on a line of round lymph-cells in the perivascular canal; it is quite distinguishable from any appearance produced by foldings or localised thickenings of the vessel tunics, in that certain segments of the canal shew a double or triple line of these bodies, and others a rupture of the sheath and a discharge of them into the space around. Arteries with these localised thickenings are elsewhere to be seen. In spite of these hypertrophic changes, vascular and interstitial, the bulk of the nerve is reduced by reason of the atrophic changes in the nerve tubules. These are closely compressed, cannot be distinctly defined, and show a coarsely granular appearance. These signs of degeneration are present in all the bundles in the central portions of the nerve, towards the periphery are less marked, and at the latter part the nerve tubules are generally swollen, varicose and ill-defined.

The Sciatic and Anterior Crural Nerves—were examined on

both sides and all showed changes of a similar kind. They have a spongier and less resistant feel than that of healthy specimens, and this is seen under the microscope to be due to the large amount of adipose tissue existing throughout the nerve, widely separating the groups of nerve bundles. The epineurium is invaded almost universally by this fat increase; in parts, its proper structure is practically replaced by fat-cells, everywhere the latter are present in it to some extent. When connective tissue-cells can be made out, they are seen to be distended with oil globules. The endoneurium has undergone marked hyperplasic change; it appears in the form of coarse, wavy, deep-stained bands of fibrous tissue spreading across the nerve bundles and obscuring many. The larger tubules, in all the nerve-bundles, seemed to have escaped change, those suffering being the smallest. Nor are the alterations at all uniform throughout the section of the nerve. Many bundles appear but little affected, whilst, in others, especially at the periphery, a very marked wasting of most of the fibres has taken place, the area of the funicle being composed of deeply-stained fibrous tissues and the remains of atrophied tubules, dotted towards the marginal portions mostly, with clearly defined uncoloured fibres. Rather seldom are seen very small bundles of atrophied tubules situated in the midst of adipose tissue, presumably the remnants of larger and healthy ones.

The vascular increase through the nerve is large. The arteries are possessed of remarkably thickened coats in which no detail of structure can as a rule be made out; they are both very numerous and much enlarged; are either engorged with blood corpuscles or blocked by a mass of welded endothelial cells.

Brain.—The skull-cap is symmetrical, rather over the average thickness; very dense, hard and heavy. The dura mater is very loose and baggy over the frontal regions; somewhat congested and pretty firmly adherent to the general cranial surface; its appearance otherwise normal. It is free from any cohesion to the inner meninges, except along the vertex at the sites of Pacchionian bodies. The superior longitudinal sinus contained throughout pale, firm, clot, the lateral dark soft clot. The subdural space contained some excess of thin, reddish, slightly turbid serous fluid.

The brain on removal appears of fair size, but narrowed in the frontal regions; its consistence is much, generally reduced, and placed upon the table; the hemispheres separate widely, uncovering much of the cerebellum.

The arachnoid over the fissures and sulci is buoyed up by fluid between it and the pia; over the summits of the gyri, closely applied. It is thickened and a milky opalescence is observable more or less uniformly, but especially along the course of the vessels. The pia was of increased consistence and hyperæmic. The membranes as a whole are increased in thickness and toughness, and strip in sheets from the brain. Their removal, performed with great care, is accomplished in spite of the diminished consistence of the cortex without its laceration, except at certain sites of adhesion to be detailed. Here the surface is left eroded in narrow patches, presenting a characteristic worm-eaten look; for the most part in the midline of the summits of the gyri, else placed a little laterally; always however at their free surface. The cortex in the sulci is perfectly smooth. The sites of erosion are the following: about the middle of the superior and middle frontal, the inferior extremities of third and ascending frontal, and the median extremity of the latter, all on the left side. On the right, the erosion is much slighter and more superficial, and appeared in two or three scattered patches over the first and second frontal gyri.

On removal of the membranes and consequently of their supporting influence, the softened brain loses all its contour and becomes flabby and flattened out, and its manipulation is far from easy. The convolutions are considerably atrophied, especially the frontal, and the intervening sulci gape widely. The gyri are of good average complexity and normal arrangement.

The basal vessels present no abnormality of structure or disposition. The grey cortex is lessened in depth, pale, and indifferently striate. The white substance much softened, œdematous and tenacious; its puncta very few. The basal ganglia are pale, look ill-nourished, and their vessels very coarse and gaping; neither on the right nor left side is any focal lesion present, nor any trace of such. The ventricles

are somewhat distended with reddish fluid; their ependyma appears smooth. The optic nerves and tracts, crura, pons, and medulla, are reserved with portions of the cortex.

Total weight of brain, 1310 grammes; right hemisphere, 550 grms.; left do., 445; cerebellum, 160 grms.; pons and medulla, 30; cerebrospinal fluid, ʒ iv.

Cortex Cerebri: Frontal gyri.—The sections prepared by the fresh method appear to have been made at the site of decortication only, since the outermost layer of the cortex is not preserved. The second and third laminæ show changes, more marked in the latter by reason of the larger size and wider separation of its nerve-cells, of the following nature. There is a large increase in the number of the free nuclei, (this existing through all the strata); obscuration of the ganglionic cells by round bodies (lymphoid?) which lay both outside and over them; a substitution of two, three or more elements similar in appearance, for the angular nucleus in the pyramidal cells, and lastly a distortion of the shape of many corpuscles owing to atrophy and apolarity. These various conditions contributed an appearance of crowding, irregularity and indefinity to the ganglionic formation.

The apolarity of so many cells is most probably not merely an effect of uneven section, but an actual loss of processes, since these lie amongst other corpuscles whose poles pursue a long course in the field. It is also noticeable that those cells preserving their polarity, retain almost invariably an intact angular nucleus, whilst those in which the latter is replaced by several rounded elements are they whose processes are most often lacking. Moreover it is observable that the former class are very seldom surrounded by the nuclear bodies, so that the latter would seem to be associated with the condition productive of atrophic changes in the cell and segmentation of its nucleus. These rounded elements are often seen in the pyramidal cell formation of senile brains, but under ordinary circumstances not in such number as are here present in this patient; and this, in conjunction with his age, indicates their import.

In no stratum of the cortex is this condition of obscuration of the cell by nuclear elements so marked as in the claustral. The large number of the corpuscles are entirely concealed by

grape-like clusters of them, from which, sometimes, a short process projects like a stalk. Of this formation the cells less affected seem to be those whose axis is vertical.

The increase in the number of vessels in this layer is perceptible; their walls are closely studded with nuclei, and often they appear to be represented by lines of aggregate elements of the same nature. Here and there, bud-like excrescences of these project from them, and not infrequently is there evidence of an active migration of leucocytes.

In the hardened sections, care was taken to cut through the layers of the cortex in the vicinity of the eroded portion as through those of the latter. The first layer preserved in these specimens shows a notable increase of the neuroglia both in the form of fibrils which give a fine mesh-work appearance to the ground-structure, and of young spider cells. The latter are numerous, and are present in no other stratum of the cortex save the first.

The vessel coats are thickened, tortuous, and wavy; the lumen of the vessel is often choked with epithelial débris. The perivascular space contains rounded, granular, or pigmented bodies, or nucleated endothelial cells. These products are occasionally seen in less amount in the subadventitial space itself. In a few instances the arterial tunics are seen lying shrivelled up in a space occupied by a compressing mass of welded endothelial cells.

Commentary.

The symptoms of Tabes were so well marked in this patient that any doubt as to the nature of the case is not possible. Those of the mental disease are however not so defined, and will need further discussion.

Before considering this subject, there are some points to be touched on in the pathology of the disease worthy of mention.

With reference, firstly, to the upward continuation through the medulla of the morbid changes in the cord. At the uppermost part of the latter, the sclerosis was most pronounced in Goll's column and the funiculus gracilis, in accordance with this, has suffered mostly. The disease in the root zone is

represented, though to a less extent, in the respiratory fascicle; and the ascending root of the 5th was degenerated. The unilateral involvement of Clarke's vesicular column in the mid and upper dorsal region of the vagal nucleus and the cell-group in the grey matter at the inferior planes of the medulla, which probably represent it, help to draw inference not only as to mutual relationship, but as regards function, the gastric disturbances being considered.

The predominance of the changes in the grey matter and cell-groups from the dorsal region upwards on the left side is interesting.

The appearance of the cord in some respects indicated perhaps a congenital defect of organisation; were this so, it might account for the more ready succumbing of the tissues of that side to disease.

The descending fibres of the fifth nerve were unilaterally (on the left) and partially affected; their possible relationship to early diplopia has been pointed out by Dr. Ross. It will be remembered that in the present case the latter affection was said to have been present for some time prior to admission, and that the fibres are diseased on the side of the larger pupil.

The bilateral mydriasis present when the patient first came under observation, may be perhaps connected with the sclerosis in the cervical region, since sensory stimuli failed to arouse further dilatation.

Accommodatory movements were not moreover affected at this time.

Later on, in the progress of the case, the ocular symptoms were, entire loss of light reflex in the left eye, partial in the right, consensual and accommodatory movements being preserved. The consensual reflex in both eyes demands the intactness of one optic tract, of the commissural fibres between the oculo-motor nuclei, and of those portions of the latter, at least, which subserve the function of light reflex. Since vision was preserved in both eyes, at any rate not seriously damaged, the optic tract must be allowed to be open for impressions.

Again, since consensual movements were present in both eyes, the light reflex portion of the oculo-motor nuclei cannot

have been seriously involved. The remaining tract is that which completes the reflex arc, a function whose morphological correlatives are supposed to be Meynert's fibres in the quadrigeminal region.

Such parts of these as could be seen in the latter did not appear diseased. Only for a limited portion of their course however were they observable. The superficial and partly the deeper medullary layers were extensively broken up by vessels on the left side, the right was also, but to a less extent, the sufferer by a vascular increase. The condition of the consensual reflex is not unfortunately noted after this just quoted stage in the ocular troubles, the loss of accommodative contraction being alone commented on. But from the pretty general affection of the oculo-motor nuclei it could hardly have been preserved. An interesting and valuable case is reported by Dr. Ross, in "BRAIN" (April 1886), in which a definite lesion of these fibres which travel betwixt the anterior tubercles and oculo-motor nucleus was discovered. Though owing to the diseased condition of the tissue in this region, my specimens were not sufficiently distinct to shew this, or such a lesion as would have produced the same effects, yet the morbid signs would have rendered the preservation of the function of the fibres quite as remarkable as its destruction.

In any case the maintenance of consensual reflex still further localised the disease.

The irregularity of the pupils suggests a disturbance of sympathetic innervation or of the influence of the meningo-encephalic adhesion existing over the middle portion of the second frontal gyrus. In connection with this we may note the fact, that the convulsive twitchings mainly affected the right side, or that opposite to the site of adhesion and the dilated pupil; and that in the intervals of actual convulsive seizures, a constant retraction of the mouth occurred with occasional sharp jerking of the head to the right side. A progressive invasion of the ocular-motor nuclei is indicated by the loss of contraction with accommodation, and the inco-ordinate movements on convergence efforts. After the convulsions, were ptosis and external strabismus on the left side. The

commencing loss of accommodative contraction is noted at the same time as the advent of the lightning pains; that of light reaction with the blunting of cutaneous reflex. Regarding the lesion of the acoustic nerve and nucleus there is but little to say. It is probable that the condition of such parts of the receptive mechanism as were involved, would likely influence, in a structural or functional modification, the receptive centre. The patient never complained of deficient or anomalous perceptions of sound, nor was the presence of aural hallucinations ever suspected. It is however just possible that such may occasionally have existed in some form, from the impulsive nature of some of his acts. He would suddenly abuse his fellow patients and the attendants without cause, and often strike at the former, always, though, choosing the aged or helpless patients. His frequently and plausibly urged excuse was that they had insulted him or called him names.

It is remarkable, when the extensive lesion of the lumbar cord is considered, that there was no coarse impairment of sensibility, which there was not, since the patient was perfectly able to recognise the nature of the substance on which he was standing, and to localise pin-pricks, etc.

Allusion may briefly be made to the greater wasting and decortication of the left hemisphere and the limitation of the latter to the frontal regions on both sides, in connection with the type of the symptoms. Also to the occurrence of vasomotor paretic signs and elevation of temperature on the side of the convulsions.

There is so much difference of opinion regarding the true nature of Locomotor Ataxy, systematic or neural, diffuse or perineural, that a short discursion as to the validity of certain observations, brought forward in evidence of a definite origin may not need apology.

A fact of great importance in the estimation of tabes as a systematic disease, is adduced by certain authors (Strumpell, Westphal), viz. the peculiar localisation of the degeneration in certain areas of the posterior columns.

That such localisation may be made in the earliest stages of the disease is probable, but that they are proof of a system

fibre affection, I do not see sufficient evidence to accept at once.

A difficulty in the investigation of the lesion in the initial periods of the disease, lies in the rareness with which a pathological examination can be made at that date. Without wishing to assume any identity or the reverse between the cord lesions in tabes, and those in cases of general paralysis with ataxic symptoms, I have nevertheless taken advantage of the opportunity such cases offer for the early observation of the degeneration of what nature it may be, in order (as one of the many objects of such a research) to discern any especial localisation to some defined area of the lesion, on its commencement. I have examined sections from the various regions of the cord in eight cases, and in the majority, the degeneration has been in a very early stage, and allowed of its characteristics being easily defined. I quote, very briefly, only such results as directly bear upon the question of primary localisation now treated of. Two prominent pathological facts in all the cases at once appear. They are a primary increase in the perineural tissue and a multiplication and enlargement of the vessels seen. Of the precedence both have to any nerve fibre affection the histological characters leave no doubt. I do not intend to enter at any length at all in this paper into the mutual relationships existent between vascular and neurogliar increase, but it may be said briefly that the vessels served as lines or centres for the development of the tissue around them. This was obvious in the case of the capillary vessels; in the larger trunks, as might be expected, not so invariably.

Of the vascular development, the following was noticeable; the enlargement of the postero-internal and external arteries, the occupation of the posterior median fissure by a considerable, sometimes very large vessel, and the presence of a branch of this also of fair size which penetrated Goll's column and traversed its midline from behind, forwards, through half its antero-posterior length. It was around this vessel that a definite excess of the neuroglia over the general hyperplasia was first seen. From this, it spread laterally, meeting a later developed growth around the postero-internal and septal arteries, until the whole column was invaded, whilst a similar

increase was taking place in the area of the internal radicular fibres.

In one instance the middle third of both columns was the richest in connective tissue increase, apparently determined around the capillary terminations of the largely developed arteries running in from the cord periphery.

The posterior commissural zones were markedly involved in the neurogliar increase, and in two cases the latter appeared by its degree to have commenced there. Only in one was there a decided overgrowth of the pia round the cord.

The summary of a more attentive investigation than the foregoing outlines represent, seems to indicate a general increase of peritubular tissue and of vessels, as a stage precedent to any neural affection, and the localisation very frequently of the neurogliar excess around the arteries. Also an especial tendency to the involving of the posterior commissural zones.

It therefore seems to me that a special distribution in the one case, and a diffuse and impartial one in the other, is not to be taken as of much account in defining the systematic character of the tabes.

When research shall have settled the nature of the lesion in this disease as neural or perineural, an advance, and obviously a very important one, will have been made towards our knowledge of its etiology.

Before I had noticed Dr. Buzzard's papers in "BRAIN," in which he lays emphasis on the frequency of a vascular origin for tabes, the prominence of the vessel changes in the present case had not failed to arouse my attention, and their nature renders it impossible to disregard the conviction, that some very important part is played by them in connection with the development of the disease. It will be recalled that their distribution is in a large number of instances the centre for an increased growth of connective tissue, for the disintegration of the grey matter or cell-groups, that their coats show signs of inflammatory alterations, and constant indications exist of an active migration of lymph-cells and occasionally of the conversion of the latter into neuroglia-corpuscles. The condition of the investing membranes did not, however, lend any support

to a theory of the origin in them of inflammatory action which might have spread in along the arteries.

Adamkiewicz has pleaded forcibly for a primary connective-tissue increase in tabes, determined around the course of the blood-vessels, and his statement lends strong support to Buzzard's views.

There is yet another pathological feature of interest occurring in a tract of close anatomical and functional relationship to the vessels and which demands especial notice, viz. the perivascular sheath and its contents. The changes in this are most obvious in the cord from the lower dorsal segment, upwards. In the regions below this the absence of the distended spaces is probably explicable by the universal sclerotic contraction, which not only obliterates the perivascular channel, but compresses the vessel walls almost to the rendering of the lumen impervious. It is likely also that obliteration of the perivascular space may occur by proliferation of the connective elements existing between its sheath and the adventitial tunic.

It is interesting to consider the relationship between the vascular and perivascular systems and the connective tissue overgrowth, and *à propos* of such, some few remarks may be made. Since the change is of the nature of an hypertrophy a condition of excessive nutrition is probable. All prolonged states of functional activity are associated with commensurate physical changes, and demand an increased supply of nutriment which is afforded by the blood plasma exuded from the capillary walls. It is recognised that since the capillaries run in lymph-spaces, the absorption of the plasma is not directly from the vessel walls but from the perivascular canal, as is also recognised the extremely intimate relationship between the lymph channels and connective tissue. An excess of exudation from the vessels under conditions when no call is made for it by the tissues, produces well-marked changes in the elements of the latter. Such undue exosmosis may arise from increased pressure in the arteries or from a morbid change in their walls; in the latter case, their permeability being increased, the escape of the morphological elements is much facilitated. And it is probable that migrated leucocytes are active agents in tissue proliferation. Supposing, now, that

some system tract by increased functional activity demands those nutritive modifications which are needful to support a consequent increased tissue consumption, changes occur to secure a larger supply of nutrient fluid to the elements and this the latter derive in the manner before described. Under normal conditions such a freer supply when afforded is regulated by the demand, an equal balance being preserved between them, and an equable relationship maintained between the amount supplied and the effete products removed.

Some prominent causes of a disturbance in the harmony of this process at once present themselves. One may be, a condition of the vessels, general or local, inherited or acquired, causing a tendency to the occurrence in them of morbid changes. Should this exist, and circumstances be added which involve a constitutional or local impairment of nutrition, and others which require increased functional performance, disease of one or another kind of the vessel structure is likely to be developed, and one of the conditions needful for the readjustment of altered relationships is lost.

Another may be that predisposition, to which the term dyscrasia is applied, to a ready proliferation of tissue-elements, and perhaps also to the production of a specific form of effete material.

Thirdly, some state of the lymph paths, primary or derived, which by leading to vitiation of their contents, and by the blocking of their channels, to the stemming back of effete material, tends to produce a spreading of lesion along their course and to impair the vitality of the tissues which they drain.

In this way may, crudely enough, be carried back the pathological process to an origin, the exciting agent or agencies of which are yet to be found.

Concluding this portion of the subject, we are compelled to notice the pathological lesions in the brain in connection with the preceding remarks. Recalling what has been said regarding the dependence of the connective tissue hypertrophy on disturbance of nutrition and of the balance between the vascular and lymphatic systems, the presence in the brain of certain conditions of an assimilable kind to those in the cord, suggest again that the cerebral and spinal lesions are part of

a wide-spread affection of the nutrient canals. The hypertrophic changes in Deiter's cells are known particularly to depend on conditions of lymph obstruction.

With reference to the peripheral zone of degeneration which is present throughout the lumbar region, this has been treated of by Déjerine and Westphal more especially, and who are exponents of different theories of its origin. The views of the former, ascribing it to an extension from a meningitis, itself set up by the posterior sclerosis, hardly suffice for the present case, since the membranes in the lower portion of the cord where the degeneration existed in the most advanced degree, were not markedly affected, whilst in the lower cervical, where the sclerosis was much less intense they were decidedly increased both in amount and vascularity, and the marginal lesion was absent. If, as Westphal suggests, it is of the nature of a system disease, since fibres in the cord presumably undergo degeneration throughout their length, such a tract would appear to be composed of fibres whose course is interrupted. Its position in the cord negatives its identity with the cerebellar tract. With regard however to that part of it in contact with the horn, it may be recalled that the posterior portion of the lateral column originates in germ-mass identical with that of the posterior column, and that therefore its participation in a degeneration of that region is not unexpected.

Various forms of intellectual and moral aberration are met with in connection with Tabes Dorsalis, and the question of their relationship to the latter disease is a most interesting one, unfortunately better recognised than understood.

Of all varieties of insanity, general paralysis has especially suffered an association. perhaps sometimes a forced one, with this spinal disease; not alone from the quasi-tabic symptoms so often existing in general paralysis, but from the tendency so constantly manifested in the two affections to approach in the bulb.

What the connection between the cerebral and spinal disease may be, is still under discussion, but that it exists is doubtless. The frequent coexistence of degeneration not only of centrifugal but of centripetal paths in the cord occurring in general paralysis, the, rarely, observed sequence of the latter to an

apparently primary and independent spinal posterior sclerosis, and the by no means rare combination of a very constant variety of mental symptoms with tabes, all offer evidence of it.

That the disease spreads as a progressive invasive lesion along functional tracts is not however apparent. Spinal disease occurs, or at least manifests itself, secondarily to the cerebral in the majority of cases, and it does not suggest itself as travelling along centripetal tracts by continuity, in fact the knowledge of physiological laws here forbids its consideration. Moreover the earliest clinical symptoms, so far as objective signs are evidence, are not infrequently those resulting from the affection of certain bulbar nuclei, in tabes and general paralysis or of the optic nerve, and the representation of this nerve in the same portion of the hemisphere as the posterior columns of the cord and its intimate connection with the oculo-motor nucleus renders its early involvement very interesting.

Returning to the bulbar lesion it is not obvious why certain nuclei should be picked out by a degenerative process, but that it is far from a haphazard event is certain. It seems however that certain physiological groups may have an especial inherent tendency to degeneration before others, and that where an unstable condition of the nervous system exists generally, or where actual lesion in some part of it is making progress, these will succumb, independent of any direct extension. The occurrence of a disease such as glosso-labio-pharyngeal paralysis which picks out a localised group for involvement tends to support this view.

It is not improbable therefore that in a conjoined cerebral and spinal affection, the respective lesions are not necessarily consecutive as regards anatomical continuity, but arise from a common cause, a proneness to degeneration of the nervous tissue at multiple points; an instance of which may be noted in cases of simple spinal disease. The development of the lesion primarily in the lower end of the cord, has its origin, it is likely, in some fairly definite source of extrinsic irritation; that of a cerebral lesion secondary to a spinal one, suggests a possible source of excitation in the latter, which some would indicate to be by the meninges, others by the

vascular system, etc. And it is to be confessed that, when a progressive upward increase in the vascularity of the cord is apparent, together with a similar vessel development in the pons and medulla, and the vaso-motor phenomena which form a part of the symptomatology of both tabes and general paralysis are considered, the estimation of this latter view is enhanced.

Whatever be the lesion, its progress, or the department of the cerebro-spinal axis in which it starts, the theory that its predisposition is in an existing or inherent instability of the latter, and its excitation is, of functional origin, appears to be one full of strong recommendation.

The nervous system shaped and modified in its development by outgoing and all the ingoing stimuli constituting its environment, whilst dependent on them for its increasing complexity must be self reliant alone for stability. The part of the organism both structurally and functionally the highest evolved, it is that which unless possessing a proportionate vitality or stamina is like to suffer, first, dissolution. And as with the whole, so with its parts; and a functional tract which receives the most intense and complex stimuli will fall under the same law. It is obvious that as emotional expressions like pleasure and pain are largely determined in degree by the condition of reactionary power in the subject, so a long-continued or oft-repeated stimulus may become of physiological or pathological, developmental or retrograde, influence according to stability or the reverse of the nervous constitution.

The influence of heredity in the case of disease in any portion of the nervous system cannot be over-estimated. We can but faintly appreciate the transmission of the minutest details of tissue organisation from the parent to the offspring; of modes of mind engendered thus, and dependent on conditions and structural peculiarities of nerve matter, we can have hardly any conception. Certain paths are prepared, the action of certain mechanisms of nerve display facilitated, and the receptive organisation is modified for the more ready appreciation of special impressions: in such terms may we express predisposition. We may go so far as to denote the latter, as a shaping of portions of the organ to the necessity of some

particular pabulum; for the food of nerve-tissue as regards functional development is stimulus; without that there is retrogression. Just as nerve mechanisms of special sense, auditory and optic, for example, may be adapted for complex combinations of sound and colours, so other parts of the receptive organ may be developed to the more ready imbibition of certain impressions. And in a faulty constitution of nerve-tissue, whose abstract correlative is manifested in all delicate gradations of the want of control and all the recalled states constituting judgment, are the conditions which render certain objective stimuli more powerful, and subjective reaction less commensurate. Such inherited defect of organisation manifests itself in some especially defined tendency, or in a general predisposition to yield to any predominant stimulus. This latter condition is perhaps the more common and the less defined; it shows itself in feebleness in controlling the repetition of certain modes of nerve excitation, either determined by the physiological epochs of life, or chance circumstances.

When the intimate interweaving of the sexual function with the development of the organism is considered, its strong influence manifested in both sexes at certain periods of life, the intensity and variety of the sensations it gives rise to, and which are expressions of a very powerful and acute excitation of certain functional tracts, it is not possible to exclude it from a very marked causal influence in disease, an influence which, regarding the diffuseness of the natural sensations occasioned by the orgasm, the implication of the intellect and emotions for the time being, must involve the whole nervous system in the effects arising from its abuse.

There is a reticence so constantly observed in regard to the sexual relationships on the part of those from whom reliable information can alone be obtained, that statistics as to excess in this respect are perhaps doubtfully reliable. And whilst on the one hand tabes occurs frequently in men at the time when the sexual powers are vigorous and even verging towards the involutory period, yet in a class of women whose mode of life exposes them to a very constant indulgence of the animal passions, tabes is not prevalent. But this is by no means a

more weighty evidence against sexual excess as influencing the production of tabes than it would be in the case of the closely related disease, general paralysis; for we must admit that the occurrence of a special disease more frequently in persons of one sex indicates something beyond a mere difference in degree of exposure to exciting causes.

The influence of heredity in our patient is sufficiently marked. As related in the history, the father was a most intemperate man, subject to paroxysmal attacks of passion; the mother, an asylum inmate suffering from chronic melancholia.

This heredity is apparent in the very dissolute habits of his life during adolescence, and a proneness to affections of both arterial and nervous structures may be considered inherent. For in the children of drunkards are frequent organic imperfections, early arrests of intellectual and moral development, and predisposition to inflammatory lesions; in them is especially apparent undue precocity, soon succeeded by retrograde changes.

Passing on to the consideration of the mental symptoms, as already observed, those in connection with tabes may assume various forms. At times they closely resemble those formerly regarded as being typical of general paralysis, and might even in such cases claim to be ranked under this head, were it not for the absence of certain motor characteristics of the latter affection, and, what is quite as important for the verification of the diagnosis, of definite pathological lesions.

General paralysis is a disease essentially compounded of two factors, the psychical and motor, but exists in so many, now recognised, diverse forms of the first, combined severally with the characteristics of the second, that it is the last which really constitute the disease as general paralysis. Separate factors, psychic and motor, but essentially coexistent.

Dwelling on the first of these for a moment, that the moral and intellectual spheres are affected differently both in time and degree is noticeable, and though both more often suffer deterioration, yet occasionally either is involved to a predominating extent; to use a metaphor, forming the heaviest weight in the balance the beam of which represents the psychical

characteristics. Hence it may be said, that on the circumstances which shape the development of some special variety of insanity, independent of the somatic element, rests the determination of the psychical portion of the disease. Alcohol, syphilis, constitutional influences, sexual excess, may thus be the modelling agents in many cases.

Though, as has been said, general paralysis demands the presence of both such factors, their development is by no means commensurate, and one may far outstrip the other in its course. In remissions is this so, apparently to the complete disappearance of the mental with maintenance or even progression of the motor. Such features have an especial association with cases in which heredity is marked. (*Mendel.*)

If we then may accept that a paramount moral or intellectual deterioration combined with certain characteristic motor anomalies and pathological lesions constitutes the disease general paralysis, we incline to class the present case as one of such a category. It is probable that but for the untimely end by convulsions, symptoms more in accordance with the usual course of the affection might have developed.

It is in no way, however, desirable to force, as there may be a tendency to do, a similarity between tabetic insanity and general paralysis. In fact a connection between the cord affection and the latter disease is perhaps better brought into view, by cases which present signs of an intermediate nature. Indeed it is not the question of the connection of the mental symptoms of tabes and general paralysis, but of the relationship existent between the cord and brain. For though the mental aspect of the first named disease has been mostly alluded to, it is by no means single as a spinal affection in having cerebral complications, and the importance of this circumstance in the estimation of certain of these as being lesions involving both encephalic and spinal systems is obvious. Paralysis agitans and miliary sclerosis are forms of spinal disease which may here be cited. In each, too, as in tabes, the variety of psychic symptoms observed most often, is that of melancholia with delusions of persecution and hallucinatory disturbances. Nor is the reason of this depressed type of mental symptomatology an obscure one. The emotional state of the individual,

whether sane or insane, is very largely the sum of the impressions derived from extrinsic and intrinsic sources; and their results are determined by the capability of judgment and control which exists. When these fail, are lost those means which enable the sane man to allocate, more or less correctly, effects to causes, by experience or inference. Blood and tissue alterations of a morbid nature in the sane and insane alike, provided the receptiveness of impressions is not too far inert, exert influence, though confessedly variable, over the emotional being.

It is difficult to imagine any disease more likely than a chronic, incurable, and advancing spinal lesion to influence with depression the whole mood of the patient's life, and when in the wide spread of the affection, the intellect is involved, and rational interpretations of his ills become distorted into a belief in them as the effects of machinations by known or unknown persecutors, as the results of loathsome disease or other cause, it is little wonder that the mental features are as some observers have stated them often to be.

Possibly, too, this depressed type of symptoms points, to a certain extent, to an etiology, in sexual excess, alcohol, and syphilis. The two latter especially of all, tend to produce a vitiation of nerve-tissue and an intellectual and moral depravation, and it is interesting to note in the three diseases, general paralysis, alcohol, and syphilis, that a lesion which may be suggestive of return to a lower type, meningeal adhesion occurs. Concluding these considerations on the nature of the mental condition in this case, in support of the view of general paralysis as existing, it is to be mentioned, that by some authors the predominance of a depressed type of symptomatology is urged in this disease: and also that it is, though rare, by no means unknown at so early a period of life, as that of our patient.

LITERATURE.

MARIE: "Disseminate Sclerosis and Perivascular Inflammation."—'Progrès Médical,' April 12, 1884.

SCHULTZ: "Multiple Sclerosis and General Paralysis."—'Arch. f. Psych.,' Bd. xi. p. 216.

PARANT: "Mental Features of Paralysis Agitans and Miliary Sclerosis."—'Annales Médico-Psych.,' July 1883.

CLAUS: "Multiple Cerebro-spinal Sclerosis."—'BRAIN,' Abstract, Vol. II. p. 142.

SEGUIN: "Anomalous Course of Posterior Spinal Sclerosis."—'Arch. de Médecine,' October 1884.

CAMUSET: "Depression in General Paralysis."—'Annales Médico-Psych.,' May 1883.

GOLDSMITH: "Early Symptoms of General Paralysis."—'Arch. of Medicine,' August 1883.

MILLS: "Locomotor Ataxia and General Paralysis."—'Journal of Nervous and Mental Disease,' July 1883.

ROSS: "Locomotor Ataxia."—'BRAIN,' Vol. IX. p. 24.

MICKLE: "General Paralysis following Locomotor Ataxia."—'Lancet,' January 1881.

MICKLE: "On General Paralysis of the Insane."

DEMANGE: "Spinal Sclerosis of Vascular Origin."—'Revue de Médecine,' Oct. 1884.

BUZZARD: "Posterior Sclerosis consecutive to Disease of Blood-vessels."—'BRAIN,' Vol. V. p. 34, and Vol. VI. p. 461.

STEPHAN: "Etiology of Tabes Dorsalis."—Critical Digest, 'BRAIN,' Vol. IX. p. 414.

ORMEROD: "Hereditary Ataxia."—Critical Digest, 'BRAIN,' Vol. VII. p. 105.

PAGE: "Peripheral Origin for Tabes."—'BRAIN,' Vol. VI. p. 361.

DÉJÉRINE: "Peripheral Nerve-lesions."—'Comptes Rendus,' 1885.

BALLET AND MARIE: "Associated Sclerosis."—'Arch. of Neurology,' Jan. 1884.

WESTPHAL: "Combined Disease of Spinal Tracts."—'Arch. f. Psych.' viii. and ix.

ORMEROD: "Associated Sclerosis."—'BRAIN,' Vol. VIII.

STRÜMPELL: "Zur pathologischen Anatomie der Tabes Dorsalis."—'Archiv f. Psych.,' vol. xii.

ORMEROD: "Cases of Spinal Disease."—'St Bartholomew's Hospital Reports,' 1878.

EXPLANATION OF THE PLATE.

Fig. 1, 2, 3.—Diseased vessels in upper dorsal, cervical and upper lumbar regions of cord.

Fig. 4.—Fibres of Stratum Zonale—(*a*) swollen tortuous axis.

Fig. 5.—Longitudinal section of Artery from Posterior Columns. Granular masses on the intima.

Fig. 6.—Section of part of the Postero-internal Artery in Cervical region.

Fig. 7.—Semi-diagrammatic sketch of the transverse section of an Artery in the Cervical segment, surrounded by large granular bodies.

Fig.1.

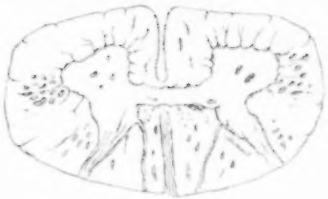


Fig.2.

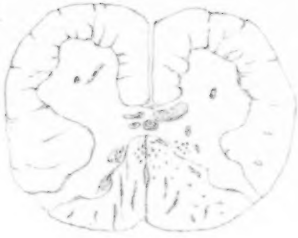


Fig.3.

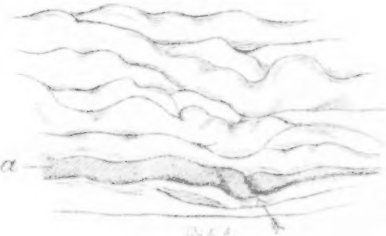


Fig.4.

Fig.5.

Fig.6.

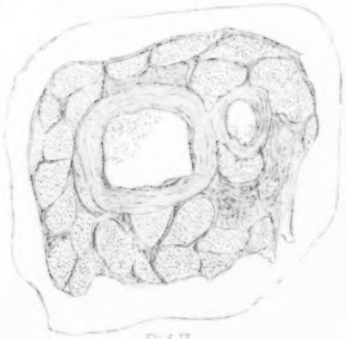


Fig.7.

FSJB del.

Danielsson & Co. lith.

PSEUDO-SCLEROSIS.

BY ROBERT MAGUIRE, M.D., M.R.C.P.

Physician to Out-Patients and Joint Lecturer on Pathology, St. Mary's Hospital.

In March of 1887, Wm. B., aged 49, presented himself in my out-patient room. The patient was stout; the face was pale except for dark-coloured blotches on the cheeks. The skin had a greasy and unhealthy appearance; the hair was grey, but plentiful. The patient walked with an unsteady gait, the legs shaking as they were moved. When he was sitting up, the head and neck showed a continuous movement, which bowed the head downwards, and twisted the chin to the right, the occiput to the left. This movement, although continuous, was not regular in rhythm, or in extent. If the head were lightly supported, it almost, but not quite, disappeared; if the patient lay down so as to be entirely at rest, no movement was perceptible. The slightest attempt at voluntary movement of the head, or voluntary support of the head, or an attempt at speech, provoked an exaggerated and more rapid movement—always, however, of the same kind, and in the same direction. The lips were very tremulous when the patient spoke, but quiet at other times. The voice was shaky, the syllables were markedly scanned, pronounced slowly, and somewhat drawled; the words, however, were distinct, the pitch of the voice was monotonous. The hands and arms showed a general tremor even when the patient was at rest; afterwards, when the patient was admitted into hospital, it was noticed that a slight amount of this tremor persisted during sleep. The tremor was slow, not very fine, and not purposeful; the fingers had no special tremor, but moved with the whole hand, and even with the forearm. The tremor was greatly exaggerated when voluntary movement was attempted. The

patient could not carry a cup to his mouth without spilling the contents; on attempting to do so, the involuntary movements, which were somewhat increased when the cup was held at arm's length, became more violent and a little more rapid, as the cup was brought nearer the mouth, until, when the cup almost touched the lips, the movement of the hand and of the head, which was brought down to meet it, were so great, that nearly the whole of the fluid in the cup was spilled before it could be got into the mouth. It was noted, however, that the movements were not at all choreic in character, and the hand was throughout carried in the desired direction, albeit in shaky fashion. The tremor was equally marked in both hands. When at rest, no tremor was observed in the legs; but when the patient attempted to rise, the muscles of the thigh were seen to shake, and the trunk was for a time unsteady; there was no sign of ataxia in the gait.

The grasp of the hands was feeble, and a general weakness was found on examining the principal muscles individually. There was no loss of sensation; there was no muscular atrophy.

The knee-jerks were greatly exaggerated; slight ankle-clonus could be elicited on the right, but not on the left side. The reflexes at the wrist and elbow were excessive, and jaw-reflex was also obtained. The superficial reflexes were normal.

The patient was somewhat lachrymose, but usually the expression of the face was apathetic; the mental state was childish. He complained of headache within an area about the size of the palm of the hand, at the top of the head. The pupils were dilated, and reacted very slowly both to light and accommodation; there was no nystagmus, no strabismus, and no diplopia. The ocular movements were carried out slowly, but this seemed to be from the stupid mental state rather than from inability to move the muscles. The right optic disc was white, the edges not well marked; but the blood-vessels were distinct; the left optic disc was irregular in form, but otherwise normal. No sign of optic neuritis was discernible; smell, taste, and hearing were normal.

The tongue was tremulous when protruded. It was red, scarred, bald and glossy in some places, covered with thick

white flakes in others. The appetite was good, and thirst was complained of; the abdominal organs were normal. In the lungs, signs of emphysema were observed. The pulse was large, soft, regular and full, and beat 68 times in the minute.

There was some pain on passing urine, and the stream was small; the urine was amber-coloured, clear, sp. gr. 1028, faintly acid, contained neither albumen nor sugar, and deposited phosphates on standing.

The temperature continued normal throughout his stay in the hospital.

The patient was born in London, but had lived most of his life in other parts of the country, and served in India and the West Indies for a time as a soldier. He was never intemperate, and retired from the army after fifteen years' service with a small pension and several good-conduct badges. Latterly he had been very badly off, thinly clad, and short of food, and had slept out in the streets for three nights during the fortnight before admission. The reason of his poverty was that the tremor of his hands prevented him getting work.

The family history was of no importance. He had suffered from gonorrhœa and syphilis, the latter 22 years ago. He was attacked by yellow fever in the West Indies; after this his hair turned white, and has remained so. He did not think he was treated with mercury for the syphilis, and certainly was never salivated.

The patient first noticed that he had a twitching at the back of the neck, with sometimes trembling of the hands, in 1876 (when he was nearly forty years of age). It gradually became worse, until in 1878 the medical staff of the Royal Marine Artillery discharged him as unfit for service. The disease progressed, and the patient stated that first the neck and head, then the arms and then the hands, were affected. Only about five months before admission had the legs commenced to be affected, and caused him to walk unsteadily. He had at different times applied for treatment at various hospitals, and been more or less relieved.

The patient was kept at rest in bed, and was given iodide of potassium in 10-grain doses, with a draught of bromide of potassium and chloral hydrate at bedtime.

On March 30th he seemed much better. The tremors were of much less extent, the sleep quieter, and the headache less. On April 14th the step was quite steady, very little shaking of the head could be perceived, and the lips were no longer tremulous; the speech had lost its shaky character, but was still slow, monotonous, and somewhat scanned. No tremor of the hands was now observed when at rest, and but little when voluntary motion was attempted. The patient was able to feed himself and drink liquids without spilling them. The knee-jerks were lively, but there was no ankle clonus. The mental state was still childish, but the patient was no longer so emotional as when admitted. On April the 21st he went to a convalescent home for six weeks, and I heard from him whilst there to the effect that tremor had almost entirely left him.

I have since been informed that some time after leaving the Convalescent Home he was admitted into the Union Infirmary, with a return of symptoms resembling those for which he was treated in St. Mary's. This time, however, he was relieved to a certain extent without the use of iodide of potassium.

Naturally in this case the diagnosis was perplexing. One thought in the first place that the extreme hardships which the patient had had of late to undergo might have produced the symptoms. It is occasionally seen that simple malnutrition of the nervous centres from starvation will cause tremor, and excessive reflexes, but the history in this case was clear that the tremor had induced the hardships, and in consequence of it the patient had been dismissed from the army. Mercurial tremor may produce an assemblage of symptoms very like that described, but this cause was carefully eliminated. Although the case was lacking in many of the symptoms of disseminated sclerosis, and although some of the symptoms found are not met with in the latter disorder, yet this was the disease which was most forcibly suggested on examining the patient. The persistence of the tremor resembled that of paralysis agitans, but the tremor in this disorder is finer, purposeful, does not affect the head and neck muscles, and moreover does not show the exaggeration on voluntary movement described

above. On the other hand, a diagnosis of disseminated sclerosis did not seem to be altogether permissible. As mentioned above, the symptomatology did not entirely correspond with that of disseminated sclerosis, but nevertheless in the latter disorder the symptoms in different cases are variable, within somewhat wide limits. The age of the patient when the first symptom appeared was very late for the commencement of disseminated sclerosis, and also we should have expected after twelve years a much more pronounced degree of the disorder at the time when he came under observation. Yet these are only probabilities. Disseminated sclerosis may, in rare instances, first show its symptoms at a very late period of life. Strümpell, for instance,[1] mentions that he has observed a case in a man 60 years of age, and confirmed his diagnosis by an autopsy. Again, disseminated sclerosis may sometimes pursue a very latent course. Nevertheless, balancing the probabilities of the whole case, I diagnosed, not disseminated sclerosis, but a functional disorder simulating it. This I think was confirmed by the progress of the case. It is difficult to believe that marked symptoms due to organic lesion could show such rapid amelioration as was here observed. In the earlier stages of disseminated sclerosis more or less improvement may be sometimes observed, but after twelve years' duration of the symptoms we could hardly consider any patient to be in an early stage. Syphilis we know may simulate the symptoms of general paralysis of the insane, and yet the recovery of the patient marks the difference between the two affections. Thinking of this I was at first inclined in the case reported to ascribe to syphilis the rôle of producing the symptoms, although, I believed, without the presence of a local lesion. For this, however, there was no real ground, and the second recovery, of which I have heard, without the use of iodide of potassium, would be opposed to such a view.

Westphal in 1883[2] described two cases in which during life many of the symptoms of disseminated cerebro-spinal sclerosis were present, and yet after death no lesion whatever was found. For purposes of comparison, I may be permitted

[1] Specielle Pathologie und Therapie IIa., p. 189.

[2] Archiv f. Psychiatrie, xiv. p. 87.

to give a condensed abstract of the exhaustive reports on these two cases.

The duration of the disease was in the first case nine, and in the second ten years from the appearance of the first symptoms. In both cases there was motor weakness of numerous muscles of the limbs and trunk, sometimes with spastic phenomena. In both cases the movements of the head were slow, tremulous, and interfered with by involuntary movements of the neck muscles. The gait was spastic-paralytic; the spastic affection was almost entirely confined to the muscles of the lower limbs; passive movements met with more or less resistance, and there was usually tremor on voluntary movement. In the upper extremities there was motor weakness, and also marked tremor on voluntary movement. In both cases the knee-jerks were present, and also Westphal's paradoxical contraction. Sensory symptoms were almost entirely absent in both cases, and the bladder and rectum were unaffected. In the first case the speech resembled that of bulbar paralysis in not being sharply articulated; but in the second case it was more scanning, without any affection of the sharpness of articulation; in both it was monotonous.

There was no nystagmus or paresis of the eye muscles, although in one case double vision had been complained of before the patient came under observation. The movements of the eyes were, however, very slow, and performed with difficulty. This symptom, Westphal remarks, is occasionally found in paralysis agitans, when the muscles become stiff, but has not been observed in multiple sclerosis. The expression of the face was stupid, the face-muscles in one of the cases trembled during speech.

Headache and dizziness were complained of, and in both cases there were psychic disorders. In the first case there was general intellectual weakness; in the second a childish manner and excessive irritability. In this latter case curious attacks occurred in which consciousness was lost, and hemiplegia of the right half of the body without affection of the facial muscles followed. Other paralytic phenomena of both sides with diminution of sensibility were observed later on.

The course of both cases was extremely chronic; the symptoms occasionally showing marked and rapid improvement. Death in the second case occurred suddenly in the night, and in the first resulted from a scorbutic condition.

At the autopsies, no change whatever, neither in the central nervous system, nor in the peripheric nerves, could be discovered in the body of either patient.

There are, it is clear, many points of difference between these cases reported by Westphal and my own, yet there are also many points of resemblance. All three presented many symptoms of disseminated sclerosis, and from a perusal of Westphal's paper, I take it that he did not wish to describe these two cases as illustrative types of a new disease. To quote the conclusion with which Westphal ends his paper: "There is a general neurosis, which one may possibly, if not very happily term pseudo-sclerosis, and which can be separated clinically from disseminated sclerosis, neither by its symptoms nor its course." I have therefore ventured to place my case in this category, seeing that it much resembles disseminated sclerosis in its symptoms, and yet, for reasons which I have already fully detailed, it seems to me probable that no organic lesion exists. Naturally, however, the criticism will be made —and, I admit, justly—that in the absence of an autopsy such a diagnosis is very uncertain.

ON SPEEDY RECOVERY FROM THE EFFECTS OF CEREBRAL EMBOLISM.

BY J. S. BRISTOWE, M.D., LL.D., F.R.S.

ABSOLUTE recovery from the effects of embolic blocking of any of the larger arteries of the brain is, for reasons which are well understood, scarcely to be hoped for in any case that comes under treatment, and must certainly be of very rare occurrence. With respect to the similar obstruction of any of the cerebral arterioles, which supply only minute districts, the conditions are different; inasmuch as, although these vessels are still without anastomoses, and serious, though limited disturbance of circulation must necessarily be the immediate consequence of the accident, it is possible that the circulation may be restored and maintained efficiently, through capillary connection with adjoining arterial districts.

The cases which follow exemplify, I believe, both of these occurrences.

In the first case, the patient, who had been under my care on and off for years with heart-disease, the result of rheumatism, was attacked suddenly with the usual symptoms of obstruction of the left middle cerebral artery, namely right hemiplegia and aphasia. Looking to the facts, there could be no reasonable doubt that her paralysis was due to embolism; and the medical man who was called in took this view, and formed an unfavourable prognosis. But she recovered perfectly in the course of a few hours, and remained free from all trace of paralysis for over four months; at the end of which time she had another attack, presumably due to re-obstruction of the same artery, which left her permanently crippled. I do not see how anything save an embolus can have caused her first seizure; and the only suggestion I can offer to explain her recovery is that the embolus was a loose

friable mass, which, after it became lodged, broke down into minute fragments, and was thus dispersed.

CASE 1.—*Heart-disease from rheumatism: followed by temporary right-sided hemiplegia and aphasia, and four months later by permanent right-sided hemiplegia with temporary aphasia.*

A. L., a married woman, aged 25, was admitted on the 11th of January, 1887. She first had rheumatism some seven years ago; and since then has been several times in the hospital under my care for heart-disease, and has frequently also come to see me as an out-patient. During all this period she has had a double mitral murmur: and from time to time has suffered from dropsy and hæmoptysis, from which, with rest, she has each time recovered.

About four months ago she came to me, apparently in her ordinary state of health, and suffering neither from dropsy nor from spitting of blood, to get my advice in reference to some symptoms which she had lately experienced. The following is the account she gave of herself. About a fortnight previously, her husband, whose occupation compels him to get up very early in the morning, went to his work as usual at about four o'clock, leaving her in bed. Between seven and eight o'clock she rose, feeling fairly well; but almost immediately afterwards fell down, paralysed on the right side and unable to speak. She lay helpless on the floor for half-an-hour or more, until the landlady of the house in which she lodged, attracted by her moaning, came to the room. A medical man was at once fetched, who recognised that she had right-sided hemiplegia with aphasia, assumed it was due to embolism, and foretold an unfavourable issue. The right arm and leg were absolutely powerless, the mouth was drawn to the left, and she was quite unable to utter any articulate sound. But she retained her consciousness. The symptoms continued without change for three or four hours, and then subsided. And on the occasion of her visit to me there remained no trace whatever of paralysis, her speech was perfect, and she was (as stated above) apparently in her usual state of health. The medical man referred to subsequently, in a letter, confirmed the substantial truth of the above narrative.

She continued in her usual health until a week before admission; when, on waking in the morning, she again found herself paralysed on the right side, and unable to speak. During this week her speech became almost perfectly restored; but the paralysis underwent no change.

State on admission.—A spare, interesting-looking young woman; suffering from well-marked right hemiplegia, without loss of feeling. The right arm and leg were absolutely motionless; the mouth was symmetrical when at rest, but drawn to the left when showing her teeth or smiling; the tongue was protruded to the right; and the right palpebral fissure was usually a little more open than the left. The tendon and superficial reflexes were natural. Her articulation

was not perfect; and she was a little slow at speaking, as if her words did not come quite readily. Moreover, there was an occasional slip in naming things; thus she called an ink-stand an ink-pot or ink-can. For the most part, however, her words were correctly applied. The heart's apex beat in the sixth space, an inch outside the nipple line. Its action was somewhat irregular. There was a loud, harsh, systolic, and a well-marked præsystolic murmur, chiefly audible at the apex. In all other respects she was fairly well.

She remained in the hospital until the 17th of April; and during her stay there was up to a certain point marked improvement in her cerebral symptoms. For a few weeks she still presented slight impairment of articulation, and unreadiness in naming; but during the latter part of the time her speech became perfect. Moreover she could read: and was able (though with awkward formation of letters) to write with her left hand. The slight facial and lingual paralysis gradually disappeared. Her leg improved slowly, and before she left the hospital she could pretty readily flex and extend the hip, knee and ankle joints, but still could not move her toes, or walk. She did not regain any use of the right fingers, hand or forearm; but to some extent recovered the power of moving her arm at the shoulder joint. After she had been in the hospital about a week, the tendon-reflexes in the right arm and leg had become exaggerated with development of ankle-clonus, and the superficial reflexes had diminished; and these phenomena continued thenceforth. It may be added that on several occasions it was noticed that when the patient yawned, the right hand and its third and fourth fingers became involuntarily extended.

Occasionally during her sojourn in the hospital she suffered from symptoms referrible to the heart; and occasionally also abnormal cardiac sounds were heard which, it was thought, pointed to affection of the tricuspid valve. But no other incident calling for notice occurred.

The second case was that of a woman who was under Dr. Stone's care, and whom I had the opportunity of examining from time to time. She was admitted with mitral-valve disease of old standing; and while under observation in the hospital had sudden obstruction of the abdominal aorta and left radial artery. She presented the characteristic phenomena and consequences of such obstructions, but so far recovered as to be able after a time to leave the hospital, still with incomplete restoration of the affected limbs, yet feeling on the whole fairly well. There can be little doubt that these sudden arterial obstructions were embolic. The case is chiefly interesting for the reasons above given. I quote it, however,

on other grounds. Eight or nine days before she was discharged, and at a time when she had in great measure recovered from her more serious symptoms, she was attacked with paralysis of the left internal rectus, of the right facial nerve, and of the right external and internal recti, with dilatation of the right pupil, vomiting and fall of temperature. The fundi of the eye remained normal. Having regard to the facts, that the patient had heart-disease of rheumatic origin, and that she had recently had sudden obstruction of the aorta and of the radial, it is scarcely possible to doubt, that this last attack of paralysis was due to a minute cardiac embolus causing obstruction of some small artery, distributed to a small district of the right crus cerebri and adjoining pons. In the course of a few days all the paralytic and other symptoms due to the accident had disappeared absolutely.

Case 2.—*Mitral disease—Sudden obstruction of aorta with disappearance of pulsation from abdominal aorta, and arteries of both lower extremities, and of left forearm—Paralysis of legs: temporary suppression of urine—Partial recovery; followed by paralysis of certain muscles of eyes and right facial nerve, of short duration.*

C. M., a married woman, aged 33, was admitted under Dr. Stone's care, on the 4th of February, 1883.

She had rheumatic fever seventeen years previously, but never any second attack; she had five children, of which two have died, and the youngest is 9 years old; has been in poor health for the last eight or nine years; and has suffered latterly from dyspnœa, cough, and pain in the region of the heart.

It was for these symptoms she was admitted. And, on examination, the heart seemed little if at all enlarged or displaced; but there was a distinct præsystolic thrill, and murmur at the apex; there was no dropsy, or albuminuria, and only a little crepitation at the bases of the lungs.

During the early part of the 6th, she had some numbness in the feet, and in the afternoon complained of severe pain about the heart, and faintness. About 5 P.M. she had a sudden pain below the left ribs, and vomited immediately afterwards.

The next day the pain and faintness and sickness had ceased; but the feet were cold; there was no pulsation in the abdominal aorta or in the main arteries of the lower extremities; she could flex the knees, but could not move either the ankles or the toes; there was loss of sensation from the knees downwards; the superficial reflexes were absent, and the patellar tendon-reflex was deficient in the right lower limb, where also there was a burning gnawing sensation. Later in the day, it was discovered that there was no pulsation in the left radial. She was anxious-looking;

her pulse was 80, full; and the heart's sounds remained as before. The temperature rose to 100·2.

After this, the condition of the patient underwent very little change for some time, but on the whole improved decidedly. The heart's action was not rapid, but was inclined to be irregular; the legs remained weak, but she recovered some power over the movements of her toes, not of her ankles; sensation returned to a considerable extent, but there still remained some numbness of the feet; the patellar tendon-reflex remained absent from the right leg; no pulsation was ever detected in the abdominal aorta, but it was fancied from time to time that it could be felt in the arteries of the lower limbs, and in those of the left forearm; some superficial patches of gangrene appeared on both legs, but remained of small size. It may be added that, for a day or two after the onset of severe symptoms, the urine was almost entirely suppressed, and she suffered from drowsiness.

At the end of March her condition was much better than could have been hoped for. She looked well, was free from pain, and cheerful; and she had recovered to a considerable extent the use of her legs and left arm; but the former were still weak, the pulsation in the formerly obstructed vessels was very feeble and often undiscernible, and the cardiac sounds and action were in the same state as when she was admitted.

On the 31st of March, however, the patient felt ill; and on the 1st of April she complained of diplopia. The right pupil was dilated; there was weakness of both external recti, of right internal rectus, and also of right facial. She had vomited once; and her temperature had sunk to 95.

On the 2nd the pupils were equal, but the diplopia continued, the weakness now being apparently limited to the right internal rectus, the left external rectus, and the right facial (which had diminished). She vomited frequently during the day. Her temperature remained low. After this, the paralytic symptoms about the face gradually disappeared; the sickness subsided; and she returned to the condition in which she was prior to the 30th of March. The eyes, examined by the ophthalmoscope, were said to be healthy; her temperature remained subnormal.

She left the hospital at her own request, on the 7th of April; the circulation in the affected limbs being still imperfect, her power over her legs still incomplete, and her ability to walk still impaired.

The next case I take to be of the same kind as the one last narrated. But the symptoms were milder and the evidence of embolism less clear.

CASE 3.—*Temporary tendency to cross paralysis of the face, due probably to embolism.*

A young medical man, who had had two attacks of acute rheumatism, of which the last had occurred eight years previously, and who had suffered ever since he was a child from megrim, consulted

me about himself in November, 1879. With the exceptions above specified he had generally enjoyed good health, and had never had syphilis. But for three or four months he had been somewhat out of sorts, and had slept badly. The day before he called upon me, while sitting in his chair, he experienced a strong sensation of warmth, with tension, on the right side of the face. He at once got up, dipped his face in water; and then, on looking at himself in the glass, discovered that he was very pale, that his sight was confused, that he had slight double vision (with, as his account seemed to show, some obliquity), and that his left pupil was dilated to the full, and inactive to light and accommodation. He was a little tottery in his walk. But he was quite himself, had no sensory disturbances, was wholly free from paralytic weakness of face or limbs, and was not aphasic. Headache referrible to the left side of the head followed. These symptoms continued during the remainder of the day; he passed a restless night; and the following morning he found that his pupils were equal and natural, and his squint was gone. When I saw him in the afternoon he still felt a little shaky; but all the other special symptoms had subsided. On examination of the heart, I detected a very slight systolic murmur audible at the apex, only at the end of a deep inspiration, and inaudible behind. I concluded that the heart was mechanically healthy; but suspected from his history and recent attack that there were a few granulations on the auricular aspect of the mitral valve. The patient is still in good health. The sensory affection of the right side of the face, combined with the dilatation of the left pupil and double vision, led me to conclude that the lesion causing his symptoms was in the neighbourhood of the pons. And from the rheumatic history, and temporary character of the attack, I attributed it to a small embolus lodged in one of the vessels of this part. The case at any rate has a close resemblance to the last. I need scarcely add that there must have been paralysis of one or more of the ocular muscles, but of which there was no evidence.

Clinical Cases.

MULTIPLE PARALYSIS OF CRANIAL NERVES—DEATH—TUMOUR OF PONS AND CEREBELLUM.

BY THOMAS BUZZARD, M.D., F.R.C.P.

Physician to the National Hospital for the Paralysed and Epileptic.

EDWARD L., æt. 40, was admitted into the National Hospital for the Paralysed and Epileptic, Queen Square, on August 31st, 1887, suffering from paralysis of various cranial nerves. Previous to admission, patient had been under the care of Dr. Ormerod.

Family and personal history unimportant. The patient had never suffered from syphilis. The following account is derived, in large measure, from notes taken by Dr. Williamson, Junior Resident Medical Officer to the Hospital.

There is paralysis of the sixth, portio dura, and portio mollis of the right side, with a little facial anæsthesia and slight weakness of right pterygoid muscles; whilst the arch of the soft palate is much reduced on the same side, the uvula deviating to the left. The right half of the palate remains motionless when patient says "Ah!"

The patient has a staggering gait. At first it seemed that this might be the result of erroneous projection due to the paralysis of the external rectus muscle of the right eye, because he walked much more steadily when this eye was shut; but latterly this has not been the case, and he has staggered, whether one eye or the other, or both were open. He complains of giddiness only when walking. He does not, however, complain of much weakness in the limbs, except as regards the legs. But it is very noteworthy that the tendon reflexes, both at the wrist and knee, on both sides of the body are enormously increased.

It seems that two years ago patient was attacked with headache in the morning, chiefly in the frontal region; and about six months later began to see double, and his wife noticed that his face was distorted. Twelve months ago he became deaf on the right side. The unsteadiness of gait has been observed for about six months. The face is paralysed on the right side, but, owing to secondary contraction of muscles, there is a *primâ facie* aspect of left-sided paralysis, the naso-labial fold being well marked on the right and absent on the left. He cannot quite close the right eye. The muscles of the right side of face react very feebly to

induced currents of moderate strength, which produce powerful contractions of those of the left side, and with a galvanic current of 5 m. a. on the right side, A S Z > K S Z.

The tongue is protruded straight. There is nothing abnormal in the condition of the vocal cords as seen by the laryngoscope.

When the patient opens his mouth, the jaw is carried a little over to his right, owing apparently to the pterygoid muscles of the right side being somewhat weaker than those of the left. It is thought, too, that the lateral movement of the jaw is stronger towards the right than the left side, and when patient closes his jaw tightly, the left masseter is more prominent than the right.

The sternomastoid and border of the trapezius may possibly be a little better marked on the left side of the neck than on the right. To induced currents of moderate strength both react equally well.

Patient can feel the touch of a feather a little better on the left side of tongue, palate, and inside of cheek than on the right, but on the lips the touch is perceived equally well on the two sides.

The deafness on the right side is absolute, and considered by Mr. Cumberbatch, who examined him, to be of central origin.

Quinine and salt-solution are perceived less distinctly on the right than the left half of the tongue.

The condition of the viscera presents no abnormality.

Under treatment with iodide of potassium and mercury the patient to a great extent lost his frontal headache, but the other symptoms remained unaltered, and in addition he began to complain of some difficulty in swallowing, the food, as he described it, tending to go to the right.

In December the patient became somewhat rapidly worse. The difficulty in swallowing either solids or fluids increased. Food would often pass into the glottis and set up coughing. On December 27th it was noted that he was unable to walk without assistance, owing to weakness of his legs, and there was a marked tendency to fall to the right, whilst the difficulty of swallowing became still worse.

On January 2nd it was noted that the pupils were equal in size, and that there was no optic neuritis.

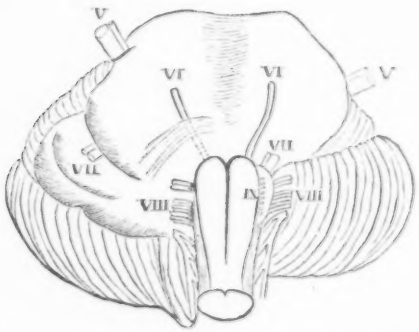
On January 4th the patient was unable to stand, and only able to raise the legs a few inches from the bed in the extended position, but could flex and extend the feet fairly well. He was also able to flex the hip and knee-joints, though feebly. The head of a pin was distinguished on the outer side of the legs and on the dorsum of the foot, but on the inner side of the legs sometimes it was not felt. A pin prick was felt as a prick in both legs. The plantar reflexes were very active on each side—and so were the knee-jerks. There was no ankle-clonus. The head of a pin was felt on both upper limbs, but rather better on the left side than on the right. He could only take liquid food. The tongue was protruded to the right. There was occasional vertical headache, but much less severe than it had been: no vomiting.

The mental state seemed dull, and he was slow in answering questions.

At the end of his dinner he suddenly fell asleep, slept heavily, and about 3 P.M. his breathing became stertorous and face livid, the skin covered with cold perspiration. His pulse was 120, fairly strong and regular. The temperature 102; pupils contracted, conjunctivæ insensitive. He could not be roused, and died shortly afterwards in a state of coma.

On post-mortem examination a new growth was found at the base of the brain. It could be separated into two halves of about equal size, the two portions being in very close apposition, and only separated by a narrow slit.

The inner portion occupied the surface of the inferior part of the right half of the pons. It measured about one inch by three-fifths of an inch. The colour was perhaps a little darker than the rest of the surface of the pons, although it bore a very strong resemblance to the rest of the brain surface. Fewer blood-vessels ramified upon it than upon the brain surface generally.



V, the trifacial nerve; VI, the abducent ocular; VII, the facial and auditory nerves; VIII, the glosso-pharyngeal, pneumo-gastric, and spinal accessory nerves; IX, the hypoglossal nerve.

Occupying the under and anterior surface of the right lobe of the cerebellum (and immediately adjacent to the just-described growth upon the pons) was a mass of precisely similar appearance, about $1\frac{1}{4}$ inch in longest diameter, and $\frac{3}{4}$ inch across. These two pieces were in close contact, only a thin slit separating them. Through the middle of this slit the right facial and auditory nerves projected, being displaced and evidently compressed by the two halves of the growth. The superficial origin of the right sixth nerve was just in front of the outer and upper angle of the growth, but not adherent to it. The posterior part of the outer (cerebellar) mass was in contact with and adherent to the eighth nerve (glossopharyngeal, vagus, and spinal accessory), but

the spinal accessory portion did not appear to be involved in the growth, which did not reach down to its superficial origin.

The medulla oblongata was pushed over to the left; the olivary body on the left side being very prominent and somewhat hard to the touch, but the appearance of the surface was normal. The olivary body of the right side appeared much smaller than that of the left, apparently from the compression to which it had been subjected.

The growth upon the cerebellum concealed the flocculus, and appeared to displace the amygdala and biventral lobe of the right side far downwards. It pressed upon the middle cerebellar peduncle of that side.

The middle lobe of the cerebellum was far out of reach of the growth, and could not apparently be affected by it, even indirectly.

This patient was exhibited to the Neurological Society on November 10th, 1887. At that time no inconsiderable doubt was felt as to the situation of the lesion, and I drew attention to the following points.

The well-marked paralysis of the facial muscles, involving as it did those of the upper as well as the lower half of the face, pointed to a lesion of the pons rather than one of the bulb, and the paralysis of the fifth and sixth nerves would also accord with a lesion in the former situation. On the other hand, what difficulty there was in swallowing at that time was not at all of a strongly marked character, and there was no such impairment of articulation as might have been expected if the lesion were in the pons. Neither was there any paralysis of limbs, and the sensation was undisturbed. The unilateral paralysis of the palate again appeared to indicate the bulb rather than the pons as the seat of lesion. The fact of the portio mollis being implicated along with the portio dura would point to lesion of nerves rather than of centre, and so would the paralysis of the sixth unaccompanied by impairment of the third. A tumour, or analogous coarse lesion, involving the middle fossa of the skull on the right side, might explain most of the symptoms; indeed, one's first thought in such a case was of a syphilitic gummatous formation in the floor of the skull. It was easy to imagine the meatus auditorius internus encroached upon, so that both the portio dura and mollis which lie in it should be subjected to destructive influence. The large superficial petrosal branch of the Vidian nerve lying in a groove on the petrous portion of the temporal bone would easily be involved in a lesion occupying this situation. If the view were taken that the nerve supply to the levator palatæ and azygos uvulæ was derived ultimately from the portio dura, through the superficial petrosal branch of the Vidian connecting this nerve above its geniculate ganglion, with the spheno-palatine ganglion, the right-side paralysis of the soft palate was at once explained. Again; the close proximity of the Gasserian ganglion and its possible implication would account for the disturbance of sensibility in the right half of the face and mouth. There was more difficulty in explaining the affection of the sixth nerve, which is separated

by the trunk of the fifth from the portio dura and mollis. It seemed hardly likely that a tumour in the middle fossa, which involved so gravely the sixth and seventh nerves, would only affect to so slight an extent the trunk of the fifth lying between them.

In these circumstances I did not feel able to express any strong opinion as to the position of the lesion, whether it were one of the floor of the skull involving the affected nerves, or a growth in the pons or bulb influencing the nuclei. The remarkable exaggeration of the tendon reflexes, however, made me incline to the latter opinion, as it seemed probable that this was due to pressure upon the pyramidal tracts.

A few weeks later the increase of difficulty in swallowing, and the growing weakness of the patient's limbs, made it unlikely that the lesion was in the floor of the skull, and the symptoms, taken altogether, appeared so conclusive, that I felt able, before the autopsy, to express the opinion that in all probability a tumour of the Pons Varolii would be found influencing the cerebellum by pressure.

The result of the autopsy leaves still undecided the vexed question of the mode of innervation of the levator palati. The facial nerve was compressed and paralysed, it is true. On the other hand, although the superficial origin of the spinal accessory appears to have escaped the encroachment of the growth, yet the medulla oblongata was pressed upon by the tumour over the whole of the olivary body. If a horizontal section be made through the bulb towards the lowest part of the olivary body, it cuts through the bulbar origin of the spinal accessory as well as the commencement of the hypoglossal nucleus. From the fact of the twist of the tongue to the right in the last part of this patient's illness, it is evident that there was paralysis of the hypoglossal on the right side. This must have been through lesion of the nucleus and not of the trunk of the nerve, for this was out of reach of the growth. *A fortiori*, it is probable that the nucleus of the spinal accessory above it was likewise subjected to damaging pressure. If the levator palati be innervated from the spinal accessory nerve, its paralysis might possibly thus receive explanation. It is right, at the same time, to remark that the vocal cords were unaffected, and that the sterno-mastoid and trapezius of the same side, although possibly, but not certainly somewhat less developed than those of the left side, showed no alteration whatever in their reaction to electrical currents. On the whole, therefore, considering the severe compression to which the portio dura was certainly subjected, and which had produced a typical paralysis of facial muscles, the evidence, such as it is, would point rather to the portio dura than to

the spinal accessory as the source of innervation of the levator palati.

Microscopical examination showed the growth to be a round-celled sarcoma. It had apparently started from two points of origin, one on the pons, the other on the cerebellum, and these in their growth had met and moulded themselves against each other, squeezing the portio dura and portio mollis between them, whilst the trunk of the fifth nerve was pressed upon and displaced upwards and forwards.

A CASE OF DOUBLE WRIST DROP APPARENTLY DUE TO MULTIPLE NEURITIS OF ALCOHOLIC ORIGIN, THE LOWER EXTREMITIES HAVING PERFECTLY RECOVERED.

BY THOMAS BUZZARD, M.D., F.R.C.P.

Physician to the National Hospital for the Paralysed and Epileptic.

JAMES M., æt. 42, engine-fitter, was admitted into the National Hospital for the Paralysed and Epileptic on January 31st, 1888, with loss of power in both hands. The following account is derived from notes taken by Dr. Williamson, Junior Resident Medical Officer.

The patient, a fairly well-nourished man, with puffiness below the lower eyelids, described how, on March 24th, 1887, he found on awaking in the morning that he was unable to use his hands. He had used them freely the day previously. The fingers were semiflexed, and patient was unable to extend them, nor could he bend his elbow. He was unable to get his hand to his mouth. He was just able to stand but not to walk. His hands felt numbed, and he could not feel when anything touched them, nor could he hold anything with them.

There was no affection of speech, no pain in the head or back. He noticed then for the first time that his arms looked thin.

He was unable to walk without help for two or three months.

He did not feel any numbness in the lower extremities. When he commenced walking he could not lift his feet, but dragged them.

The attack took place when he was on board ship off Rio Janeiro. There was no other case of illness on board, and he had not been on shore.

The hands have been gradually getting worse, and patient has been unable to do any work since the onset of paralysis. The arms, he says, have been gradually getting thinner. He had never felt any pains or rheumatism in his arms.

As regards antecedents, he had suffered from scalp wound and erysipelas of the head and face two years ago, and thinks he was unconscious for four weeks. He has had ague four times slightly. There is no specific history. He has had gout in the right big toe three times. There is no history of acute renal mischief. Many years ago he used lead in fitting joints of machinery, but not for the last six or seven years. In his trying work in the

engine-rooms of steamers in the tropics he has drunk very freely indeed. He had been taking about fourteen ounces of whisky daily, besides malt liquor for two or three years at least before his attack.

The grasp of the right hand is 25, that of the left 30. Both hands are dropped at the wrist, and the fingers semi-flexed without contraction. There is slight wasting of the thenar and hypothenar eminences, more on the right hand than on the left, and of the first interosseous space on the right side. Patient is unable to fully extend the fingers. He is able to separate and approximate the fingers of both hands, but not to the full extent.

He can only imperfectly extend the wrists, and flexion is feeble. All movements of the elbow and shoulder can be performed. He can pronate and supinate the hand. There is slight wasting of the muscles on the extensor side of the forearm.

The other muscles of the forearm are somewhat flabby, but not wasted. The supinator longus stands out on each side, when the arm is flexed at the elbow, the forearm being in a position between pronation and supination.

The wrist-jerk and triceps-jerk are present on both sides.

There is nothing wrong to be observed about the spine or lower limbs.

The patient walks well, without unsteadiness. The knee-jerks and plantar reflex are normal on both sides. There is no ankle-clonus.

There is no affection of the facial or ocular muscles, and the tongue is protruded straight. The pupils react to light and to accommodation. Symptoms of commencing albuminuric retinitis are observable in the left eye.

There is no affection of cutaneous sensibility.

No blue line is to be found on the gums. The circulatory and respiratory systems show nothing abnormal. The urine is sp. gr. 1012, alkaline. It contains albumen—no sugar. No casts are observable. The patient micturates very frequently.

Electrical examination; Galvanism.—In the forearms all muscles react readily except the extensor communis digitorum, which reacts feebly.

On the R. side, the extensor communis digitorum,	K S Z > A S Z.
On the L. „ „ „ „	K S Z = A S Z.
In all the other muscles of the forearms	K S Z > A S Z.

Faradism.—Both forearms. Muscles of thenar and hypothenar eminences, the extensor ossis metacarpi pollicis and the flexor muscles react quite readily to moderate strength. The extensor communis digitorum does not react at all to the strongest current.

The supinator longus, extensor carpi radialis longior, and brevior, and the extensor carpi ulnaris react only to strong induced currents.

Mr. Gunn reports of the eyes:—"*Left side.*—The disc is practically normal, the upper edge a little indistinct. At the yellow

spot the usual asterisk arrangement of small white spots characteristic of albuminuric retinitis. The *right* eye is normal.

"The right eye seems to have good vision, and there is no central colour scotoma. The left at first failed to detect green on central fixation, and first recognised it when it fell on the fundus a little outside the yellow spot; red was detected normally."

This patient presents superficially the aspect of a typical example of lead palsy. The history of the mode of attack, and the fact that patient has used no lead for six or seven years, and that he presents no blue line on his gums, afford practically conclusive evidence against this view of his case. And this is also confirmed to a considerable extent by the fact, that the supinator longus muscle shares with the extensors (although unequally) in the disorder.

The mode of attack, and the circumstance of his lower extremities being also affected at first, the feet not being able to be lifted but being dragged, point strongly to the probability, that he has suffered from multiple neuritis. The only apparent cause of this is the excessive indulgence in spirits which the patient freely admits. A suggestion from a speaker at the meeting of the Neurological Society at which I exhibited the patient, that it possibly might be an example of beri-beri, is negatived by the fact of the patient not having been on shore, and being the only invalid. It is not so easy to be sure that the patient is not suffering from the remains of an attack of anterior poliomyelitis. Against this view it will be noted that there appears to have been a good deal of numbness in the hands at first. The exact symmetry of the lesions again is much more in favour of polyneuritis, as is also the circumstance of the supinators being associated with the extensors of the wrists in the altered electrical reaction.

At the meeting in question, the President called attention to the huskiness of the patient's voice. Dr. Semon, who has since examined him at my request, reports as follows:—

"Both vocal cords move with respiration and phonation, but on phonation there is a curious relaxation of the left vocal cord, the inner border line of which is not straight but concave (paresis of the left internal thyro-arytenoid muscle). It is also seen, when the patient is told to produce a vowel (*a*) twice over in quick succession, that only the right cord moves energetically inwards and outwards, whilst the movements of the left are so lazy as to be almost imperceptible."

Dr. Semon is disposed to think that the laryngoscopic observation favours a diagnosis of multiple neuritis. The ophthalmic examination, although somewhat favouring the idea of chronic alcoholism, is not conclusive.

The case appears to me to be unusual, from the fact of the vastly more serious implication of the upper than the lower extremities. According to my experience, although the upper extremities are much more often than not involved, and frequently to a very serious extent, it is on the lower extremities that the severity of the attack in alcoholic neuritis appears to fall. So that in cases where the hands long continue useless it will ordinarily be found that the legs are still more powerless. In this instance the man's legs have apparently recovered perfectly, but his arms, though now improving, are quite incapacitated.

NOTE.—Since the above was in press I have seen in private practice a case of alcoholic neuritis, in which there still remain complete incapacity of one hand and partial disablement of the other, whilst the lower extremities have entirely recovered.

CASE OF ATROPHY OF THE FRONTAL AND PARIETAL LOBES OF THE BRAIN, CONSEQUENT ON LONG-CONTINUED PARALYSIS OF THE LIMBS FROM SPINAL DISEASE.

BY SEYMOUR J. SHARKEY, M.D., F.R.C.P.

S. P., æt. 37, was admitted into St. Thomas's Hospital under the care of Dr. Harley on the 2nd of August, 1884, and died there on the 3rd of February, 1887. She dated her illness from the preceding April, when she was confined of a still-born child, since which time she had suffered from a pain in the back, and had wasted and lost power in her arms and legs, to such a degree that she could neither stand, nor carry anything in her hands. On admission, her legs and right hand were quite powerless, and she had retention of urine. On the 27th of March, 1886, a year before her death, pain and loss of power were noticed in her left arm, which gradually increased. She continued absolutely confined to bed during the time which she remained alive in St. Thomas's Hospital—a period of two years and seven months. During the whole of this time her legs were completely paralysed, and they became rigid and flexed, while her right hand was affected in the same way. Her left hand and arm were partially paralysed for the last year only. Sensation was retained throughout, except that on rare occasions some dulness was said to be noticed; but this was a transitory phenomenon. She had at first retention, and then incontinence of urine, with cystitis and attacks of high fever.

During the latter part of her life she put on flesh, and her general condition improved very much. Throughout, her mind was free from disturbance, and not in any way deficient. She died on the 3rd of February, 1887.

Post-mortem examination showed that the body was very well nourished.

The legs were rigidly flexed on the abdomen and at the knees: the fingers of the right hand were also flexed.

The lungs, heart, and liver were very atrophied, but otherwise presented no signs of disease.

The spleen was the seat of amyloid changes.

In the pelvis of the left kidney were several irregularly shaped calculi with centres of uric acid, and a deposit of phosphates externally. The pelvis of the kidney was inflamed, the ureter not. Amyloid changes were found in the straight vessels and Malpighian tufts. The right kidney and its pelvis were in a similar condition.

The bladder was inflamed, and its walls thickened.

Uterus and ovaries atrophied.

The brain[1] was healthy, so far as its structure, vascular supply, &c., were concerned; but there was very marked atrophy of the frontal and parietal lobes on both sides. The convolutions were thin, and separated from their neighbours by broad sulci, full of clear fluid. The atrophy was very striking, and was most marked in the upper extremities of the two ascending parietal convolutions of the right hemisphere, and in the superior parietal lobule. The same convolutions were most affected in the left hemisphere, but, in addition, the middle third of the parietal convolutions looked strikingly reduced in size. Microscopic examination showed that there was no disease in these convolutions, but that they were simply atrophied. The frontal convolutions on both sides were in a condition of atrophy similar to that seen in the parietal, but less in degree. In both hemispheres the occipital and temporo-sphenoidal lobes stood out in marked contrast, being well developed, and presenting an almost smooth expanse of broad convolutions, with very ill-marked sulci between them. The central ganglia appeared to be healthy, and those in the right hemisphere were examined in sections and found to be normal.

The bodies of five vertebræ—the sixth and seventh cervical, and the first three dorsal—were carious, and had partially disappeared. There were no collections of pus, but as the disease was most advanced on the right side, a marked curvature, with its concavity towards the right, had been produced. This had caused a displacement of the spinal cord to the left. The dura mater in this region was thickened, but it was not adherent to the spinal cord. The latter was softened for an area of about two inches, and below that level there was well-defined lateral sclerosis in the usual position. The nerves issuing from this part of the cord did not appear to be diseased, nor were the nerve trunks or large muscles of the limbs structurally altered, so far as one could judge from naked-eye examination; but the small muscles of the hand and some of the deep muscles on the flexor surface of the forearm were atrophied and pale.

[1] See Plate, Fig. 1.

The point of special interest in this case, for the sake of which I record it, is that it illustrates the separation in the cerebral cortex of the sensory from the psycho-motor centres. We have frequent opportunities of seeing this proved, either by the results of disease or of experiments on the cortex, lesions of which, in certain regions only, produce paralysis.

But it is a very rare event to get a good specimen illustrating this connection between certain parts of the brain and voluntary muscular action by the secondary effects on the cortex cerebri of experimental or pathological lesions of the cord, nerves, or muscles. The patient, whose brain I now show, may be said to have had voluntary muscular action reduced to a minimum for two and a half years. Her legs were quite paralysed, and incapable of being affected by voluntary stimuli during all that period; so was her right hand, and the inability to use the hand made it practically useless to move such muscles of the arm as were not paralysed. The left arm and hand were only affected for one year before death, and that partially. Being kept absolutely at rest in bed for two and a half years precluded the opportunity of bringing the muscles of the trunk into play, and even the head must have been subjected to but little movement.

The special senses, on the other hand, were all good and in use, and common sensation was little if at all affected. Hence if atrophy follows disuse, we should have expected those parts of the cortex which subserve voluntary muscular action to have wasted, while those which preside over sensation ought to have remained well preserved. Now without going into detail, the striking feature of this brain is the atrophy of the frontal and parietal, and the plump condition of the temporo-sphenoidal and occipital lobes. This, I think, is in accordance with our present views as regards the localisation of the sensory and motor centres.

We know too little of the physiology of the intellectual operations to say whether one part of the brain is more concerned in them than another. But even if it be held that the frontal lobes preside more especially over the processes referred to, one would not, on that account, have necessarily looked for mental deterioration in this case. For the absence of determinations to, and execution of, voluntary motor actions, might give rise to some atrophy of the parts concerned in them, without producing serious diminution of intellectual power.

A FATAL CASE OF TUMOUR OF THE LEFT AUDITORY NERVE.

BY SEYMOUR J. SHARKEY, M.D., F.R.C.P.

On January 20th, 1887, R. G., æt. 41, came to my out-patient room at St. Thomas's Hospital, bringing with him a letter from one of the district Committees of the Charity Organisation Society, in which I was asked to say whether he should be sent to a Convalescent Home. He had already been in Bethnal Green Medical Mission Hospital for ten weeks, and after that at High Barnet Convalescent Home.

The patient informed me that up to the present illness he had been a healthy man; that he had never had syphilis.

His symptoms had first appeared during the preceding year (1886), and consisted of pains in the head, attacks of giddiness and "faintness," noise in the left ear, deafness, and some slight deterioration of sight. During the last five or six months he had lost a good deal of weight.

The headache had been in the vertex and frontal region. The giddiness was not only continuous, but increased paroxysmally, and in the paroxysms things appeared to revolve towards the left. These attacks occurred eight or nine times a day, and he was not free from giddiness even in bed. He had singing in the left ear and deafness, symptoms which had come on simultaneously.

In addition to the attacks of giddiness he suffered from what he termed "fainting fits." These attacks were ushered in by increased giddiness, but not by increase in the noise in the left ear; his sight got dim in them and he lost consciousness for some ten minutes. He never had vomiting, his appetite was good, and his bowels rather confined. His urine was free from albumen, and all his organs appeared to be healthy. On examination with the ophthalmoscope I found that he had double optic neuritis with great swelling of the discs. He had no paralytic phenomena.

Mr. Clutton, the Aural Surgeon to St. Thomas's, happened to be seeing patients in a neighbouring room, so I asked him to report on the man's condition.

His report was as follows: "Membrana tympani normal on both sides. Watch heard in right ear at two inches, in left not at all. Fork heard normally on right side, on left not at

all, whether with auditory canal open or closed. Paroxysmal increase in the noise in left ear not synchronous with attacks of vertigo; this is against the disease being aural; but the vertigo is like that which is usually looked upon as aural, being both constant and increased in paroxysms. The examination with the fork would indicate that the disease is either in the brain, in the trunk of the auditory nerve, or in its peripheral expansion in the internal ear. For if it were in the middle or external ear, conduction with the fork would be intensified."

The presence of optic neuritis enabled me, however, to localise the disease within narrower limits, by eliminating the internal ear, and I came to the conclusion that the affection was intra-cranial, involving, if not necessarily originating in, the trunk of the left auditory nerve or the auditory centre in the temporo-sphenoidal lobe. A little more consideration at the time might have enabled me to localise it definitely in the trunk of the nerve; for if the centre only had been the seat of the disease, the patient would have had deafness, but not vertigo. The patient was subsequently admitted into the hospital, Mr. Nettleship kindly taking him into the Ophthalmic ward, as the medical wards were full. He was there treated with iodide of potash and mercurial inunction. His improvement was, however, slight.

He went out, and was again admitted on May 28th, 1887, into a bed kindly put at my disposal by Dr. Stone. He was then much in the same condition, except that he was thinner, and that his sight was worse. He had no paralysis and no anæsthesia, and his knee-jerks were brisk and equal. The left pupil was slightly smaller than the right, and there was thought to be slight deviation outwards of left eye when the patient looked straight forwards.

From time to time he had "fainting fits," in which he lost consciousness but had no convulsions. On June 17th, however, he had an attack in which some shaking and general rigidity were observed.

In August he had hallucinations of vision, seeing people round him and talking to them. Then for a short period he was violent, and refused food, and had to be fed through the nose. These symptoms, however, passed off. He occasionally complained of numbness in the gums, and his fits were more constantly ushered in by twitchings of the muscles of the limbs, especially on the right side. His sight too became so bad that he could scarcely see at all, and there was very slight paresis of the left side of the face. During the first three days of November he had fits in which he was unconscious as long as an hour, and in which his breathing became stertorous, and he died on November 4th.

At the post-mortem examination all the organs were found to be healthy, and the only pathological change present was an intra-cranial new growth, the nature and connections of which were as follows:

The tumour, which was rather larger than a horse-chestnut, occupied the angle between the cerebellum and pons on the left side, and in its growth it had formed a deep, cup-shaped cavity in the left lobe of the cerebellum. At first it was not clear where the tumour had taken its origin. It was found, however, to be entirely separate from the cerebellum, and only lying loose in a depression in it, which it had formed by pressure. It had pushed back the flocculus, and had flattened very considerably the left side of the pons and medulla oblongata.

It lay in the occipital fossa, and terminated in a short nozzle, which was lodged in a cavity in the petrous part of the temporal bone. This cavity showed no signs of disease, and the bone around it was quite healthy. It was, in fact, the internal auditory meatus expanded by the pressure of the tumour. The whole internal ear, together with the ossicles, was healthy.

After hardening the brain in spirit, the nerves were carefully traced. The fifth and the sixth were found a little displaced, owing to the alteration in shape of the pons and medulla. The facial was seen rising in its ordinary position, just below the tumour, and running along its anterior border towards the petrous bone. Over the lower surface of the growth posteriorly ran a nerve, subdivided into several bundles, and taking origin in the position usually occupied by the vagus. Close to the facial, and in front of the vagus, a nerve was found, originating in the ordinary position of the auditory: it entered the tumour, and formed the only connection which the latter had with any part of the surrounding nerve structures. Moreover, on section of the nose-shaped prominence which occupied the enlarged auditory meatus, the nerve was found, under the microscope, issuing from the mass of spindle-cells of which the tumour was mainly composed. Thus there can be no room for doubting, that the tumour grew in connection with the left auditory nerve.

The drawing by Mr. Shattock, which is here reproduced,[1]

V. marks the 5th Nerve.
VI. „ „ 6th „
VII.A „ „ Facial.
VII.B „ „ Auditory.
VIII. „ „ Vagus.

[1] See Plate, Fig. 2.

gives a beautiful representation of the tumour, and its relations to the cranial nerves medulla oblongata, pons, and cerebellum.

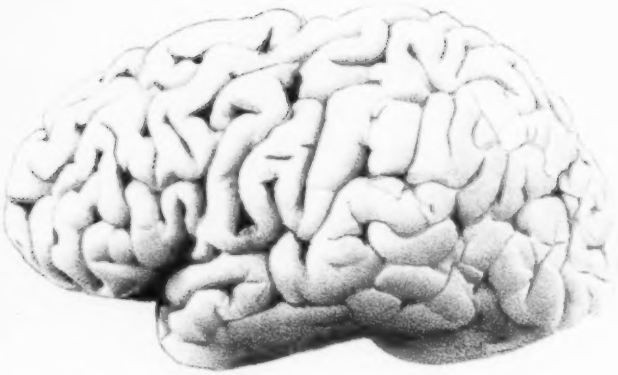
The interest of this case resides principally in the phenomena which ushered it in. The later symptoms were the secondary results of pressure on the neighbouring parts. Thus the shaking and rigidity of limbs which were observed in the epileptiform seizures which he had towards the end of the illness were due to the pressure of the tumour on the pons and medulla. The headache, hallucinations, and attacks of violence, and the optic neuritis with subsequent blindness, may occur in cerebral tumours occupying various positions.

But the very earliest symptoms of which he complained must be attributed to the disturbance of the function of that nerve alone on which the tumour was developed; and so we have a very interesting case of a pathological experiment performed on a nerve without any surgical operation, and consequently without the added disturbances which the latter may entail, and which often make the results of experiments inconclusive.

The earliest symptoms in this man's case were giddiness—continuous and paroxysmal—deafness and noise in the left ear, symptoms which are familiar to us as those of Menière's disease, in which the peripheral expansions of the auditory nerve in the labyrinth are affected. In experimental section of the auditory nerve in animals, and in destruction of the semicircular canals, the same intense giddiness has often been observed. But it has been attributed by some to the accidental lesions produced by the operation, and not to that lesion which the operators had in view. The present case, however, seems to me to be conclusive, as to the direct connection between section of the auditory nerve and the giddiness which follows it.

It has been noticed in the case of experiments that in time the giddiness and inco-ordination tend to diminish, and this was so in the case under consideration. The patient complained but little of it during the last few months of his life.

Another point of some interest is this, that the case would almost certainly have been put down as a case of Menière's disease at the time when I first saw him, unless an ophthalmoscopic examination had been made.



From a Photograph. Fig. 1.

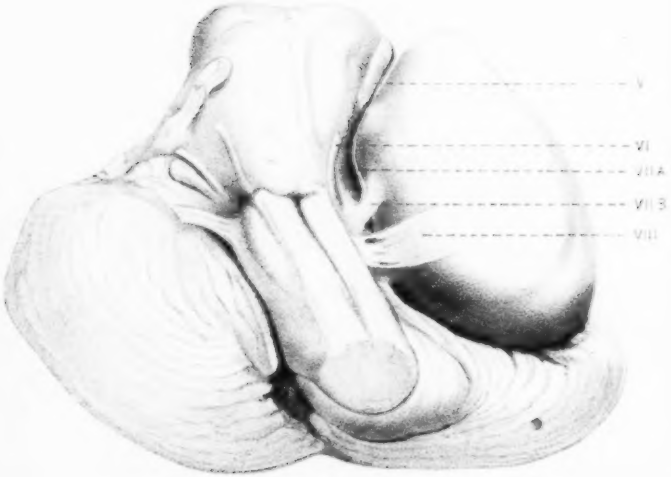


Fig 2.

S.G. Shattock del. Danielsson & Co lith.

EXPLANATION OF THE PLATE.

FIG. 1.—It is very hard to bring out in any single view, however well presented, the contrast between the Fronto-parietal and the Occipito-temporal regions which was so evident to any one who held the brain in his hands, and turned it in various directions. The plump condition of the occipito-temporal convolutions, and the well-filled spaces between them, presented appearances very different from the shrunken convolutions and the large furrows separating them, seen in the fronto-parietal region.

FIG. 2.—View of under-surface of Pons, Medulla Oblongata, Cerebellum, and Tumour.

V. represents the left 5th Nerve.
VI. " " 6th "
VIIA. " " Portio Dura.
VIIB. " " Portio Mollis
Entering the Tumour.

VIII. represents the left Pneumogastric coursing over the under-surface of the Tumour.

A CASE OF THROMBOSIS OF THE LONGITUDINAL SINUS, TOGETHER WITH THE ANTERIOR FRONTAL VEIN, CAUSING LOCALISED FOCI OF HÆMORRHAGE, WHICH PRODUCED REMARKABLY LOCALISED CORTICAL EPILEPSY.

BY VICTOR HORSLEY, B.S., F.R.S.

Surgeon to the National Hospital for Paralysed and Epileptic, &c.

THE following case I publish, because it is valuable in localising the situation in man of Dr. Ferrier's area for the turning of the head and eyes to the opposite side, and at the same time the anterior limit of the upper limb area, together with the special representation of the segments of that limb at the anterior part of the region devoted to it.

Briefly summed up, the case is as follows: A man who had been suffering from tubercular disease of the bones for some months, suddenly developed symptoms of thrombosis of the longitudinal sinus, with cortical epilepsy as a result. The exhaustion produced by the epilepsy rapidly caused death. Unfortunately I never saw the patient during life, but I performed the post-mortem examination, for which I am very greatly indebted to the kindness of my friend, Dr. Lunn, and for the opportunity to publish the case in the interests of cerebral localisation, while I am also very much obliged to Dr. Courteen for the clinical notes.

J. J., aged 24, labourer, single, admitted into St. Marylebone Infirmary, Dec. 18, 1886. There was, on admission, chronic erysipelas of the scalp, a suppurating sinus situated over the right malar bone, and another over the left parietal bone just above and in front of the parietal eminence. These were freely laid open, but others developed in their immediate neighbourhood.

History.—The patient had been abroad some years, and a few weeks before admission was said to have knocked his head against a wall.

The patient, after admission, never seemed to recover, but gradually became weaker, in spite of extra diet, tonics, and the administration of iodide of potassium. Fresh suppuration was detected and treated vigorously, but without arresting the progress of the disease. A week before his death, the patient

became much feebler and thinner, developed a hacking cough and became very drowsy.

March 4, 1887, 1.15 A.M.—The patient had a fit, followed soon afterwards by several others; each was of short duration, described by the Ward Sister as not lasting more than a minute. The fourth fit was observed by the Sister, who described it as follows: "The patient was lying on his back; first the head turned to the left, and he made a slight moaning noise; then the eyes turned upwards; he threw the left arm straight forwards, then upwards and outwards; the head then turned slightly to the right, and lastly both legs became convulsed, the mouth slightly open. Pulse 120."

The fifth fit occurred at 4.25 A.M.; but in this it was noted that in addition to the protrusion in extension of the upper limb, the left wrist was forcibly extended, or, according to the words of the Sister, "turned upwards," while the fingers were directed downwards. In fact, there was extension of the wrist with an interosseal position of the fingers. Several fits were observed after this last, but they gradually became weaker. The patient was quite unconscious from the onset of the first fit, and at 6.4 P.M. he died.

The movements observed, therefore, were first turning of the head to the left; then raising the arm at right angles to the trunk in complete extension, with extreme extension of the wrist and interosseal position of the fingers; gradual turning of the head to the right, and subsequently the rest of the body involved in the spasm.

Theoretically, we should have had to account for the particular primary movement and march of the spasm, in this case a largish lesion at the posterior border of the head and eyes area, where it becomes merged with the anterior border of the upper limb area in the right hemisphere, and then we might or might not have in addition a lesion in the left hemisphere at the highest point of the area of representation of the head and neck. According to the investigations of Dr. Beevor and myself,[1] these lesions, supposing both to exist, should have been situated in the following positions: the first, the larger, should have covered the posterior third of the middle frontal convolution, and should have to a slight extent invaded the anterior border of the ascending frontal convolution. The other smaller lesion should have been situated in the left marginal convolution in front of the vertical line carried up along the direction of the præcentral sulcus to the longitudinal sulcus. This point was also localised by Professor Schäfer and myself.[2]

As a matter of fact, both these imagined lesions were found

[1] 'Philosophical Transactions, 1887 and 1888.'

[2] 'Proceedings of the Royal Society,' 1886. 'Philosophical Transactions,' 1887.

to exist in the present case exactly in the spots posthumously diagnosed; but it is hardly necessary, perhaps, to point out that the second lesion was not, as it were, necessary for the production of the movement of turning of the head to the right towards the close of the fits, since it is well known that exhaustion of the head and eyes centre of the one side is followed by unresisted action of the centre of the opposite side, and consequently a corresponding change in position of the head. The accompanying diagram, which is outlined from a photograph of the brain very kindly taken for me by Dr. Wilson, late Senior Resident Medical Officer at the National Hospital for the Paralysed and Epileptic, Queen's Square, shows exactly the seat of the lesions.

I will now proceed to describe the conditions observed in the autopsy.

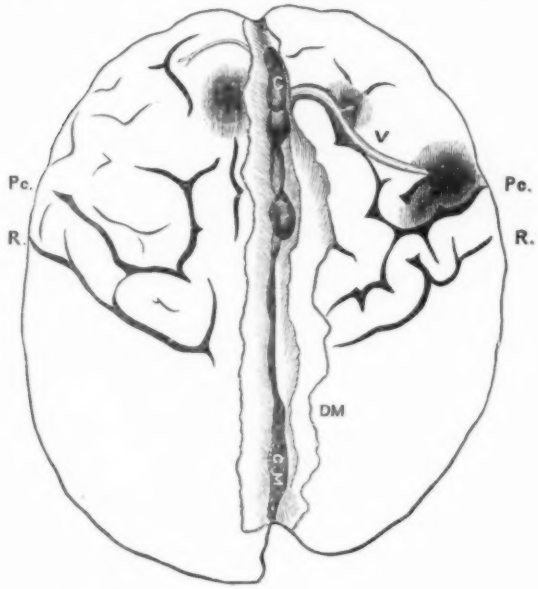
Autopsy.—Body well nourished. Rigor mortis not quite passed off. Over the right malar bone there was an old sinus leading to bare bone, and also a sinus over the left frontal bone, which similarly terminated in necrosed bone. There was a patch of caries, with destruction of the external table of the skull, midway between the posterior, superior and anterior angles of the left parietal bone; it formed an E-shaped area, the long side of which rested on the superior temporal ridge. The extremities of the area were each 2 centimetres in breadth. There was no trace of similar caries or necrosis in the rest of the skull.

On removing the calvarium, the surface of the dura mater appeared normal except over the anterior third of the longitudinal sinus, which appeared to be distended and discoloured. On the left hemisphere the dura mater was rather more prominent and bulging than on the right.

On slitting up the longitudinal sinus, there was found posteriorly a post-mortem blood-clot, partly decolorised, soft and dark black in colour for the most part, terminating anteriorly in a firm ante-mortem adherent clot, which extended from the junction of the middle and the posterior thirds of the superior frontal convolution, forwards as far as the crista galli. This clot was of a dark brownish-black colour, except opposite the middle of the superior frontal convolution, where it was very pale. The large vein on the right side which joins the sinus at this spot (referred to as the anterior frontal vein) was absolutely blocked by an ante-mortem clot, which as far as the superior frontal sulcus was of a black colour, but below that point of a pale greyish colour, and terminated below at the junction of the middle and lower thirds of the ascending frontal convolution. On the left side the veins entering the sinus from the external surface of the hemisphere were all pervious, excepting a small vein which joined the sinus opposite the junction of the anterior middle thirds of the superior frontal convolution, this small vein being blocked similarly to the large one on the right side.

This thrombosis of the sinus and veins caused the following lesions.

Right hemisphere.—The surface of the hemisphere appeared perfectly normal, except in the neighbourhood of the blocked frontal vein before described. The posterior sixth of the middle frontal convolution in its whole breadth was the seat of a hæmorrhagic extravasation of blood. The ascending frontal convolution was highly congested, especially in its anterior border; the



R.—Fissure of Rolando.
Pc.—Præcentral sulcus.
C.—Ante-mortem clot } lying in longitudinal sulcus.
C.M.—Post-mortem clot }
D.M.—Edge of Dura Mater.
V.—Thrombosed vein.

The dark patches are the seats of hæmorrhage. The Sulci over the posterior part of the hemisphere are omitted for clearness.

membranes also of the superior frontal sulcus were congested along its posterior third, and there was a slight hæmorrhagic extravasation in the outer border of the middle third and the superior frontal convolution of this side (the right).

Left hemisphere.—There was a dark black hæmorrhagic focus occupying the anterior half of the middle third of the superior frontal convolution for half its breadth. This, the only lesion

in the left hemisphere, was situated (as before described) at the highest point of the area for the head and neck in the left hemisphere. It must here be mentioned that the whole of the surface of the left hemisphere was covered with a thin, tenacious, light red fibrinous exudation, which was adherent to the inner surface of the dura mater.

The rest of the central nervous system was perfectly normal. The other organs, moreover, presented no abnormality worth noting, except the lungs, which were both thickly studded with miliary tubercles. The occurrence of venous thrombosis in the cases of tubercular disease of the bones, &c., is too well known to pathologists to need special mention here, but we need the accumulation of many cases like the present to enable us to diagnose with certainty whether the cortical epilepsy is actually caused by hæmorrhage, as in the present instance, or, as would be quite as possible, by a tubercular nodule growing in the cortex, since in the latter case we could interfere with great benefit to the patient, whereas in the former case of course such treatment would be out of the question in the present state of our knowledge.

A CASE OF CHRONIC MENINGITIS, PROBABLY SYPHILITIC, AND CAUSING PROGRESSIVE DEMENTIA.

BY FRANCIS WARNER, M.D., F.R.C.P.,

AND

FLETCHER BEACH, M.B., M.R.C.P.

WILLIS C., a boy aged 7 years, first came under observation as an out-patient at the East London Hospital for Children in January 1879.

He was considered healthy at birth, and was suckled eleven months; as an infant he suffered from snuffles, thrush and sores about the nates. He never had fits. He had been attacked with measles and whooping-cough, but got well over them, and appears to have been a strong boy. He went to school, did his lessons fairly well, and could write; he continued bright and well till about eight or nine months before he was first seen. About April 1878 his mother began to notice a certain difficulty in his movements, which continued. At the same time he began to complain of headaches, and to cry for slight causes; a sudden noise would disturb him, and the sight of certain objects or animals, or even of other children, would frighten him. He still continued at school, but the teacher said he had gone back in his learning.

When first seen he could not protrude his tongue properly; his teeth were ground, the upper incisors lost, and it was ascertained that he had the habit of tooth-grinding, especially at night. He often complained of being tired, and of having headaches; sometimes he vomited. He was not mischievous at this time. His head was well-shaped, 20¼ inches in circumference; his weight was just 3 stone. His heart and other organs appeared healthy.

To summarise the facts observed during the next six months. When sitting on the floor he had great difficulty in getting up, and could not do so without using his hands; he could go upstairs well, but had great difficulty in coming down. The calves each measured 8½ inches in circumference; the knee-jerk was well marked on either side; his gait was very stiff. He would sometimes grind his teeth during the day as well as night.

Purgatives occasionally brought away a few round worms, but their expulsion had no material effect upon his condition. Mercury with iodide of potassium was given almost continuously through the year.

In February 1879, it was noted that at times he seemed in a

dormant condition, as if mentally lost; the left pupil was usually larger than the right, but they varied in size.

In March he was reported to have had "an attack," the first that had been noticed; he trembled, and would have fallen had he not been supported; he did not lose consciousness. When seen he was stupid, and would not shake hands. Soon after this he became weaker, dull and stupid. He could speak, but had some difficulty in swallowing; he would fill his mouth without swallowing the food.

In June he vomited occasionally; he walked badly. There was distinct mental deterioration, he could hardly say the alphabet; occasionally the bowels acted involuntarily; he had grown stouter.

In July 1879 he was admitted an in-patient under Dr. Eustace Smith, to whom we are indebted for the following notes.

"A well-nourished boy, with healthy complexion. He is very troublesome, often trying to get out of bed, often shrieking, apparently neither from pain nor from passion. He talks only nonsense, neither asking for food nor for anything else. When given toys he is amused with them for a few minutes, and then ceases playing with them, but does not break them or throw them away. He can run down the ward, but does so as if frightened. The facial muscles all act well, there is no paralysis of any ocular muscle, the left pupil is larger than the right. Sight appears good, no signs of keratitis. On tickling the soles of the feet, reflex action is well marked. He is generally good-natured, but does not appear to know what he is doing; more he likes music. He often vomits and is very constipated.

"When given a picture-book, he will play with it, but keeps it upside down. At times, however, he attempts to hurt other children, and will knock and bruise himself." He was discharged as being unmanageable.

During the remainder of this year he was frequently seen; at times he was very irritable and unmanageable, and his hands became very cold and blue. Sometimes there was vomiting, but no complaint of pain. The signs of intelligence varied; in December he was taken very ill, vomiting the whole of one day, after which he lay in a condition of torpor, and for the two succeeding days was laughing, singing, and grinding his teeth.

January 19th, 1880.—He was brought to the hospital much worse. He came into the consulting-room looking dull and silly, grinding his teeth, his face and hands bluish. He could stand and walk a little; he walked to the hospital with difficulty. He was dirty in his habits, and had to be fed. He took no notice of questions put to him, and would not look at any one speaking to him, nor protrude his tongue when asked to. He could drink well. Pupils were large; the heart was very feeble. No fits. He was again admitted in-patient under Dr. Eustace Smith. Another attack occurred in the hospital, in which he was unconscious for six hours; no convulsions; at times he would scream and laugh. He was discharged as a hopeless imbecile.

In June 1880 he was admitted in-patient at the London Hospital. He was still well nourished, inclined to be noisy, said nothing but "Mamma" and "Dadda." He was constantly grinding his teeth, and never would play. He could stand, and there was no palsy of any cranial nerve; he could apparently see and hear. His eyes were frequently examined ophthalmoscopically, but no changes were seen.

In September 1880 he was admitted into Darenth Asylum, and the following note was taken: "A thin, dark-complexioned boy, aged nine years; manner peevish, with dirty habits. He can hold objects with his right hand, but not with his left, which usually has the thumb turned in; the second toe of his left foot is drawn down. He cannot stand without support, and his gait, when supported, is jerky. Pupils are sluggish, and speech has been lost. No disease of spine."

He became very weak and emaciated, and died January 19th, 1882.

The following history of the family was obtained: His maternal grandmother was epileptic. His mother, aged 43 when the patient was first seen, had been liable to spectral illusions, generally seen at night when going to sleep; she had been liable to headaches.

The father, aged 44, a sailor, had been under the care of Dr. Stephen Mackenzie, who informed us that he found signs of syphilis in the man from a sore contracted in 1851; he had a temporary attack of hemiplegia when 13 years old. There had been six offspring in the family: (1) miscarriage; (2) ditto; (3) born at eight months, then died; (4) the patient; (5) a girl, healthy; (6) a miscarriage.

Autopsy (twenty-seven hours after death).—Body much emaciated; bed-sores. On removing the skull-cap, the dura mater was seen, and this was found to be adherent to, but easily separable from, a subjacent false membrane, the surface of which was glistening, and of a pink colour. This false membrane was about two lines thick on the right side; on the left side it was thicker. It had evidently been formed some time, but the upper surface appeared to have been recent. It was attached here and there to the upper surface of the pia mater by thin membrane, but was easily separated everywhere from the brain. There was some fluid beneath the membrane. After the brain was removed, the membrane could be traced along the floor of the skull as far as the superior margin of the petrous bone, but it did not line the posterior fossæ. In front, on the right side of the floor of the skull, it was thin, but at its posterior part it averaged from 6 mm. to a quarter of an inch in thickness; on the left side it was not so thick. The upper surface of the brain was congested, and the vessels of the pia mater were injected.

The brain weighed 1 lb. 11 oz. Convolutions were not so simple in character as in cases of congenital imbecility.

The other organs were normal, except the left lung, which was collapsed from old pleurisy.

The wood-cuts of the brain are from photographs taken for me by my friend, Mr. W. T. F. M. Ingall, and represent the brain after being in spirit, and then in a weak solution of chloride of zinc for some time. They are two-thirds of its actual present size.

This case is interesting in many respects. It was clearly one of acquired imbecility, due to chronic meningitis, probably syphilitic in its origin, though prolonged mercurial treatment appeared incapable of arresting the disease.

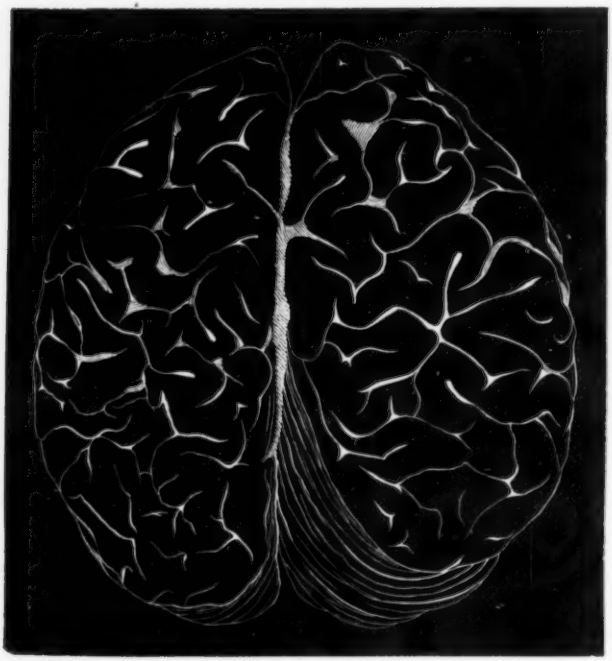


FIG. 1.

The case is representative of many in Darenth Asylum, where, owing to the influence of heredity, indicated here by the history on the maternal side, the child is born with an unstable brain, easily upset by any cause. In this case the cause was meningitis, probably due to syphilis inherited from the father. Syphilis is not a common exciting cause of imbecility, but according to Heubner, hereditary predispo-

sition to nerve-disease appears to exert an influence in determining the syphilitic poison towards the nervous system. As is usual in such cases, the brain is of comparatively small

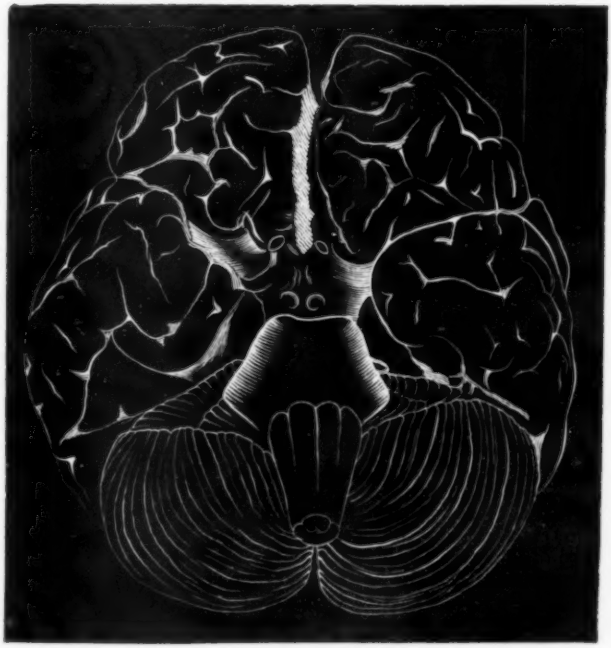


FIG. 2.

size. The determining cause of the onset of the pachymeningitis does not appear clear from the history.

The following references are given, illustrative of the connection between syphilis and idiocy and imbecility:

IRELAND: 'Idiocy and Imbecility.'
GRIESINGER: 'On Mental Diseases.'
LANCEREAUX: 'Traité de la Syphilis.'
HUGHLINGS-JACKSON: 'London Hospital Reports,' vol. i., 1864.
MAUDSLEY: 'Pathology of Mind.'
BUCKNILL AND TUKE: 'Manual of Psychological Medicine.'
MENDEL: "Ueber Hereditäre Syphilis in ihrer Einwirkung auf Entwickelung von Geisteskrankheiten."—'Archiv für Psychiatrie,' Bd. I., 1878–79.
BURY: "The Influence of Hereditary Syphilis in the Production of Idiocy or Dementia."—'BRAIN,' Vol. VI., 1883.

A CASE OF ALMOST COMPLETE ANÆSTHESIA WITH ATAXIA OF THE LIMBS.

BY C. E. BEEVOR, M.D., M.R.C.P.

Assistant Physician to the National Hospital for the Paralysed and Epileptic, and the Great Northern Hospital.

Amy S., aged 24, noticed seven years ago that she gradually became weak in the right hand and arm, and at the same time they felt numb; she frequently dropped things, and had difficulty in picking up small objects. Two months later she noticed that the right leg felt heavy and numb, and she had some difficulty in walking, and it dragged after her. One year after the onset she found out that she had lost feeling in the whole of the right half of the body. Two years from the onset the left arm began to be affected with numbness and weakness similarly to the right arm; the left leg was subsequently affected, though to a less degree than the right.

She had measles when 15 years old; she never quite recovered her health after this illness, and her present illness came on about two years later.

At different times she has had sharp shooting-pains in the right arm, lasting only for a second or two.

Since onset she has had some difficulty in holding her water.

She first came under my observation in June 1884, and she has since been an in-patient in the National Hospital for the Paralysed and Epileptic, under Dr. Hughlings-Jackson and Dr. Ferrier. At that time—three years ago—she presented the following condition:

She was a well-made girl, and though rather apathetic, she did not present any characters pointing to hysteria.

Motor system.—Face normal, no paralysis of the motor cranial nerves. Pupils equal, reacting to light and accommodation.

Right upper limb is somewhat wasted, and especially the extensors of the wrist, the deltoid and triceps muscles. She has much difficulty in separating the fingers and in extending them; the grasp is weak, but flexion of the fingers is much better than extension; she has great difficulty in picking up small objects with the right hand. Flexion of the elbow is better than extension; she can abduct the shoulder to the horizontal line, but not beyond this. She has most of the movements of the limb, but they are performed very slowly, and with much hesitation.

Left upper limb is less wasted than the right, but there is not much difference in the measurements of the forearms and the middle of the humerus of either side. The grasp is better than on the right, and all the movements are better performed, but they are slower and more hesitating than normal. She can, however, pick up a pin with the left hand.

The *Lower limbs* are well developed, and she has all the movements in them, but the left is stronger than the right leg. She is able to walk a mile with assistance.

Knee-jerks are very excessive; *ankle-clonus* very well marked on both sides. *Plantar reflexes* are normal.

Sensation is very much affected nearly over the whole body, but especially on the right side.

The right arm is anæsthetic to tactile sensation throughout, and is analgesic to painful sensations as far as the elbow; the loss of tactile sensation extends up the neck to the face—where at times she can localise correctly, but often not—and above to the middle line of the head, and behind to the spine; the trunk is anæsthetic on the right side to the middle line.

The right leg is completely anæsthetic to the knee, and to a less degree in the thigh.

The left arm is anæsthetic to the elbow, and analgesic to the wrist; the left leg is only slightly anæsthetic to the knee, while in the left trunk she can localise tactile sensations fairly well.

Ataxia and *loss of muscular sense* are very marked. She cannot stand with the eyes closed, but staggers at once, and falls.

When the eyes are closed she cannot touch the nose with either hand, but she has more difficulty with the right than with the left upper limb.

She has no idea, with the eyes closed, when the right hand or elbow are moved passively, or when the left fingers and wrist are flexed, but she can just tell when the right shoulder and the left elbow and wrist are similarly treated; she cannot tell the position in which the right forearm or the left hand is placed. With the eyes shut she cannot appreciate different weights put into the hands.

In the legs she cannot appreciate movements, or the resulting position when the right foot and knee are bent, or when the left foot is moved.

The muscles of both forearms react normally to the faradic current.

Sight normal; no colour-blindness; field of vision not affected. Optic discs normal.

The above account refers to her condition in 1884.

Present condition has not altered much, but the anæsthesia is more marked than previously.

Right hand is rather more wasted, and has a tendency to become "clawed," and she is unable to separate the fingers; it takes her some time to extend and flex the fingers, as if she had to overcome some obstacle.

Sensation to touch and pain is almost completely absent over the whole of the right side.

The left arm is anæsthetic to the middle of humerus.

The left leg is completely anæsthetic, but she can feel painful impressions. In lying down she can only feel the left half of the bed, and it seems as if she were lying on the edge of the bed, and would fall out towards the right side. When sitting down she cannot sometimes feel the chair at all, and with the eyes closed she seems to be floating in the air.

Electric stimuli are very imperfectly felt on the right side of the body.

She has the same degree of power whether the eyes are open or closed, and she can kick out with either leg equally well with eyes open or shut.

Plantar reflex is now absent on both sides.

The *knee-jerks* are excessive, and *ankle-clonus* is present on both sides.

In the above case the chief points are:—Almost complete anæsthesia of the right half of the body and limbs, and a considerable amount of anæsthesia of the limbs of the left side and to a less degree of the left half of the body; loss of muscular sense as regards position and the appreciation of weights, together with marked ataxia in all four limbs, when the eyes are closed. Associated with these symptoms there is very little paralysis.

She is able to walk a mile leaning on another person's arm, and although she has lost the finer movements of the right hand, and she has some difficulty in moving the other joints of the right arm, there is no marked paralysis.

There is ankle-clonus and excessive knee-jerks on both sides, but there is no rigidity of the lower limbs.

The case is somewhat similar to those referred to by Dr. Bastian in his paper on "The Muscular Sense" read before the Neurological Society last December, and especially the case of Remigius Lens, published by Spaeth.[1]

The diagnosis of the case is difficult, the symptoms are rather like those of hysteria; but she has been in the hospital twice as an in-patient, and all treatment has failed to produce any improvement.

[1] Beitr. zur Lehre von der Tabes Dorsalis." Tübingen, 1864.

TWO CASES OF INSANITY DEPENDING ON SYPHILITIC DISEASE OF THE ARTERIES.

BY GEO. H. SAVAGE, M.D.

I HAVE ventured to record the following cases as very good examples of two very different classes of cases which are met with in asylums; in both there was no doubt about the history of syphilis, and there is no doubt about the changes which were found after death being such as have been usually attributed to that disease.

In the first case, progressive dementia associated with local softenings in the brain was present; while in the second, the disease of the arteries was followed by the special form of degeneration which we call general paralysis of the insane.

Why in one case the process should be one of simple decay, while in the other it was associated with a period of excitement, and why with this progressive disease there was a temporary lull, I am unable to explain.

No form of treatment is of the least use when arteries have become as diseased as they were in both these cases, and the history of syphilis in no way made the prognosis better.

In both cases there was a distinct history of nervous instability in the families.

Syphilitic Dementia with localised Softening, with marked Disease of Arteries.

Notes before admission.—M. W. Single. Worked for living as a clerk. He belongs to a noted family which has produced distinguished men as well as spendthrifts. A cousin married a notorious woman, who had a child, which child deprived this man of the inheritance. He was an odd boy at school, where it is said he drank. He contracted syphilis at the age of 17; had no serious secondary symptoms. He led a solitary life, and drank very heavily till about four years ago (1883), when he reformed. He had a fit about two years ago (1885), which left him with right-side hemiplegia and aphasia. He regained power in his leg and arm, but his speech has been defective ever since. He was in no way more peculiar than usual till November 1887, when he began to fancy things which were not true. He would lie in bed by day, and at night he would wander round the square

where he lived. He forgot his appointments and neglected his duties. Dr. Savage saw him Dec. 16th, 1887, and was told there was some suspicion he was malingering because some women had accused him of having relations with a child below the legal age. He was said to have been given of late to the lowest female company. He fancied he was in debt, and had hallucinations of smell.

On December 17th he was admitted to the hospital.

On admission.—Paternal first cousin was insane. Father died of asthma and thrombosis.

Patient was eccentric, a good linguist and well educated; always melancholy, and avoided outdoor exercise. Drank hard from 1875 to 1879. Paralysis two years ago supposed to be due to drink. Right arm lost power. Speech affected. Had syphilis when about 17 (now 37). Has been abstainer for four years.

Time of earliest symptoms about three weeks before admission. He was silent and melancholy, and said he was in debt, which was not true. He wandered about, became more silent, refused food, saying it was poisoned and that his teeth were all gone. He would not wash, and said he had attempted his life. He said his coffee and food were unpleasant.

Tongue clean, pale; no tremor. Appetite bad. Wet and dirty. Urine 1020, acid, phosphates, no albumen. Pupils equal dilated, reacting to light.

After admission.—Tall; dark brown hair and beard; grey eyes; face sallow; tongue clean, no tremor; depressed anxious expression. Refused to answer any question; was restive and inclined to be violent while being questioned. Restless during examination of lungs, but would not speak.

Dec. 19, 1887.—Sits unoccupied. Takes food badly. Wet and dirty. Says he cannot talk. Sleeps badly.

Dec. 23*rd.*—Fell down in the gallery this morning. He was apparently conscious, and had not had a fit, but looked faint and ill. He was found to have lost power, and there was anæsthesia of the left hand. He formerly had right hemiplegia and aphasia.

Dec. 24*th*, at 6 P.M.—Pulse feeble; loss of power in left arm; eyes turned to the right. Anæsthesia of left side, and marked coldness compared with the other. Breathing stertorous; fed with nose tube.

Dec. 25*th.*—No change, except that the right hand is kept clenched, and there is ankle-clonus of the left ankle.

Dec. 26*th.*—Pulse feeble; tongue and lips dry and foul; eyes directed to right. Right hand clenched. Anæsthesia of the whole of left side. Temperature of both sides equal, 99°. Fed with nose tube. Pot. iod. gr. x, liq. hyd. perchlor. ♏ 60, t. d. s.

Jan. 2*nd*, 1888.—Some improvement; more colour in face; breathing normal; eyes still turned to right, but takes hold of a proffered hand with his right, which is less firmly clenched, and can be opened voluntarily. Some ecchymosis in the outer canthus of the left eye. Mouth very dry; right half of tongue thickly furred; left half clean. Distinct pallor of left optic disc.

Jan. 4th—Not so well; breathing again stertorous; pulse small and rapid; bowels acting frequently; motions very offensive. 11 P.M.—Was found cold and almost pulseless, but rallied gradually.

Jan. 5th.—Evidently dying. Pulse feeble; surface cold; bowels acting constantly. Died at 9.30 P.M. Shortly before death he had some clonic spasms of the left side.

POST-MORTEM.—*Sixty-two hours after death. Weather mild.*

Body fairly nourished; rigor mortis in legs; no bed-sores or marks of injury. Brain, 46 oz.; heart, 14 oz.; right lung, 40 oz.; left lung, 30 oz.; liver, 59 oz.; kidneys, R. 6 oz. and 5 oz.; spleen, 6 oz.

Calvaria thick; dura mater apparently normal; slight excess of sub-arachnoid fluid. P. M. peels readily; no sign of gummatous infiltration at base. In left internal carotid is a long, recent, loose clot.

In right middle cerebral artery, about an inch from its origin, is the characteristic appearance of syphilitic arteritis, the vessel (or rather two parallel vessels) being impervious at this point. The vessels of the opposite side appear to be normal. No ætheroma seen. On making a section of the brain the puncta cruenta are very marked, and just outside the frontal end of the caudate nucleus, and before reaching the level of the ventricles, there is seen to be on the right side a spotted patch. On cutting down there is found to be a small cavity, the size of a bean, situated at this spot, and containing some grumous fluid. The walls appear to be smooth, as if the cavity were not quite recent. The surrounding white matter is somewhat soft and pulpy. On the left side there is seen to be an old cavity with smooth walls, apparently the result of a former softening. This occupies the internal capsule just outside the caudate nucleus at the level of the anterior end of the optic thalamus. There is no sign of any hæmorrhagic condition, and no blood pigment about. Rest of brain apparently normal.

Both lungs congested and œdematous; right hypostatic consolidation at base; heart pale and flabby; valves normal; kidneys large and pale; spleen and liver normal.

The above case may serve as a clinical contribution to the localisation of function, and also as a typical case of mental breakdown associated with syphilitic disease of the arteries. The patient belonged to a highly nervous family. He contracted syphilis when about seventeen, and did not suffer from the disease of the arteries for eighteen or twenty years. One of the earlier of the marked symptoms of degeneration was exhibited by excessive sexual desire, or loss of control of this desire. He fell into very low company, and was threatened with trouble, in consequence of frequenting the society of very young girls.

On first seeing the patient, it was difficult to decide whether he was malingering to escape from justice, and but for the traces of the aphasia, with the history of the fit, I should have been in doubt. As has been recorded, he had a second fit, which proved to be the beginning of the end.

There were two local softenings, one which accounted for the right hemiplegia and the aphasia; the other, of more recent origin, was in front of the corpus striatum—in fact, in the area which Dr. Hale White has pointed out as possibly being associated with the control of the body temperature. In this case there was, after the loss of power on the left side, a marked change in the temperature on the left side; but, beside this, the left half of tongue was cleaner and moister than the corresponding right half. There was pallor, too, of the left disc.

The innumerable local wastings which may result from syphilitic arteritis make the careful recording of such cases of service toward the elucidation of the specific function and relationships of the parts of the brain which depend on local blood supply, so that such cases may enable us to separate distinct areas of blood supply, and compare them, with special areas of functional activity or irritability.

Geo. S. R., admitted, Oct. 16th 1886.

Maternal grandmother epileptic. Mother poisoned herself at time of climacteric, had previously suffered from metrorrhagia, followed by anæmia. One paternal aunt died of phthisis.

Patient's History.—Unusually brilliant. Athletic in Cambridge eleven. Sober. Had syphilis while at College. Slight secondaries. Slight sunstroke some years ago. Disappointment at not gaining an appointment in July.

Earliest symptoms, end of July last.—Sleeplessness following shock. Given large doses of chloral and opium, for about a month, during which time he had two or three attacks of coma lasting about two days (thirty hours). Loss of memory and of nerve power; excitement and violence at times.

On examination;—Tongue flabby, tremulous, slightly furred; appetite good; bowels regular; lungs normal; heart normal; reproductive organs normal. Eyes good, pupils large, react to light; right larger than left. Ears and hearing good.

Common sensibility good; says he can't feel; temperature normal; reaction to questions slow or absent; depression; memory, recent and remote, confused. Sleep disturbed, dreams not marked. Walk wanting in firmness and elasticity, pigeon-toed; reflexes very brisk; knee-jerks.

Dynamometer, R. 65, L. 53.

A tall, somewhat slim, fairly well-nourished man. His hair, beard, moustache and whiskers are black and coarse, with here and there a grey hair. His complexion is fresh and ruddy; his eyes

dark brown, with widely dilated pupils, the right pupil being slightly the larger. His aspect is somewhat vacant. He goes about with half-open mouth and elevated eyebrows. His tongue and lips are tremulous, his speech explosive and blurred. Both hands are somewhat blue and cold. When turning suddenly, he staggers; his walk is feeble and pigeon-toed; reflexes brisk. Seems very confused; cannot say how long he has been here, how he came, or where he is. Is quiet; occasionally plays chess, but as a rule is unoccupied. Takes his food well, but is a little restless at night. Has a tendency to collect rubbish. Uses the spittoons to pass water into. Does not show any tendency to suicide or violence. His memory for recent and remote events is confused; he does not answer questions readily. One cannot get any evidence of hallucinations, but he has a delusion that he is watched by a policeman. The patient, in short, appears to be suffering from general paralysis of the insane.

Oct. 17th.—Believes that the night watchmen are policemen who were sent to watch him. Still in a quiet stupid condition.

Oct. 20th.—No change; moved from No. 1A to No. 3 Infirmary.

Oct. 27th.—A little brighter; answers questions better. Still unoccupied and quiet. Recognises his wife. Circulation better.

Oct. 31st.—Quiet and unoccupied, but looks much brighter; less taciturn.

Nov. 9th.—Memory bad; thinks he has been in Bethlem six weeks. Quiet and rather apathetic; reflexes brisk; right pupil larger than left.

Dec. 8th.—Leave of absence three weeks. No marked mental improvement since last note.

Dec. 29th.—Extension of leave, three weeks.

Jan. 3rd, 1887.—From his father's letter of this date it appears he still entertains the delusion of his wife's unfaithfulness, and he hears voices, otherwise he appears in good health.

Jan. 19th.—Extension of leave three weeks.

Feb. 9th. Ditto. Ditto.

March 2nd.—Letter from patient's father announcing his complete recovery. Discharged well.

July 17th.—Re-admitted.—After leaving as above he kept well till May, when he was in Greece. He had intermittent fever a month before, and suffered from headache. In May he left his wife in Corfu, and went to Athens. On the way he was excited. He was away from his friends a week. On the previous Sunday he had made many mistakes in reading the lessons, but preached a very good sermon. He said he was worth millions, that he was captain of the vessel, and Christ. He recognised his wife when brought home, but was very violent. Was always attacking his attendants. He had a great contempt for the Greeks, and used to fight them on every opportunity. They were all very frightened of him. He used to say that an Englishman should be equal to twenty Greeks, and he tried to demonstrate this on every occasion.

He said he would ask God to hang him. He never showed any

tendency to suicide. He attacked every one who came near him most violently. Always liked to be with his wife.

Before the last attack he thought a book was lying on the table, when it was really at the end of the room.

Had prickly feeling in arm and leg. Had difficulty in opening his jaw when first beginning to eat. Tongue furred, flabby. Lungs and heart normal. Eyes normal, pupils equal. Reaction to questions; not to the point; voluble, profane. Maniacal. Restless. Walk natural.

On admission.—Tall, dark, powerfully made man. Black eyebrows and beard. Very violent at times, constantly talking. His talk consists mostly of obscenities, with biblical quotations. He seized Mr. J. round the neck, and said he would hug him, and proceeded to illustrate his words. He collects scraps of paper, leaves, sticks, and stones, and fills his mouth with them as well. If interfered with, he spits at every one within reach. He occasionally adopts an ecclesiastical tone of voice, and speaks of damnable heresies, love of the girls, mixing in Greek and Italian words.

He says he has great wealth. Whilst coming from Corfu he wanted to get out, to show that he could walk on the sea.

July 19th.—Still violent, destructive and mischievous.

July 21st.—Says this is the most excellently managed hotel he was ever in. He has gloves on, and requires an attendant to constantly watch him.

July 23rd.—Very violent. Says a voice tells him to die. "If you wait a minute you will hear the voice. There it is! a million pounds for this man!"

July 28th.—Violent.

August 2nd.—Tore up his bed last night. Talking constantly to himself and swearing. In side-arm dress.

Aug. 5th.—Up in airing court; quieter.

Aug. 6th.—Much quieter; says he is going to India.

Aug. 8th.—Says he is the "Bishop of Chester and Hawarden." "Many thanks for getting me the appointment. It is worth a million a year. You know I did not seduce that girl, I married her, the daughter of the Governor-General of Cyprus. It was all a mistake, you know I would not do such a thing." 3.15 P.M. Hyoscyamine, gr. $\frac{1}{60}$. Was very quiet while it was given, and afterwards.

Aug. 9th.—Says he felt much better after the injection. "Slept for two years and a half."

Aug. 12th.—Very noisy when spoken to. Began to cry, and says he never seduced that girl.

Aug. 15th.—Hyoscyamine, gr. $\frac{1}{60}$. Was restless after it, and lavish in his curses to the —— —— who gave it him.

Aug. 16th.—Anxious not to have another injection, it made him feel so ill.

Aug. 19th.—Has been tearing up and chewing his bedding. When asked why he did so, he said, "It is locusts and wild honey. A fellow who does not eat locusts and wild honey when he can get it, must be a d— fool."

Aug. 22*nd.*—Vomiting several times this morning. Began before breakfast to go to bed. Much bile vomited.

Aug. 23*rd.*—Not sick to-day. Much quieter. Conjunctivæ rather yellow.

Aug. 24*th.*—He vomited once to-day after dinner, but seemed better later in the day.

Sept. 1*st.*—This morning he was walking about in the airing court, talking much as usual. Said he felt quite well, and made some of his usual statements about having "married the girl." He looked much thinner than when he came in, but otherwise seemed fairly well. At 4.30 P.M. he was sitting in the gallery with a newspaper before him. About 5 o'clock he was noticed to slip off the seat he was sitting on; he became unconscious; no cry. Was then put on the bed; his left arm became drawn up, and his left leg straight, but twitching. The right arm and leg were flaccid; face not affected. Breathing stertorous, temp. 98·4. Later breathing became very irregular and stertorous. He had several attacks of congestion of face, with blueness of lips and tongue and stoppage of breathing. These attacks ran into one another without definite convulsions. He was unable to swallow, and mucus collected in the back of his pharynx and seemed to choke him. It improved the breathing when he was turned on his side and the mucus wiped out of his mouth. About 7 o'clock his left arm was flaccid, and it was then found his left leg was cold, and right hot. Temp., R. axilla, 103. Injectn. Æther-hypoderm. ♏ 30 at 9.5. At 9.30 the eyes were examined, and the only thing noticed was congestion of retinal veins.

He remained in an unconscious state, with occasional stoppage of pulse and breathing, the breathing being slow, 10 to a minute, and stertorous. He died at 11.35 P.M.

POST-MORTEM.—24 *hours after death, weather cool.*

Rigor mortis in legs, not in arms. P. M. lividity on back. No bed-sores or bruises. Encephalon, 50 oz.; heart, 12 oz.; liver, 7 oz. Lungs, R. 27 oz., L. 26 oz. Kidneys, R. 5½ oz., L. 6 oz. Spleen, 4 oz.

Calvaria irregular in thickness. In region of middle meningeal arteries on both sides the channels for the vessels, especially the small ones, are very deep, and in some instances tunnels. Attachments of superior longitudinal sinus very prominent. Dura mater somewhat adherent to bone over both parietal eminences and surface of frontal region. Arachnoid rather thick and opaque; no excess of fluid. Pia mater somewhat thick, but peels fairly easily, except over each superior frontal convolution, where there is some adhesion. Some congestion of veins at occipital region. No marked wasting of convolutions on surface. Left vertebral artery very much smaller than right. At commencement of basilar artery is a round projection of the vessel about the size of a small pea. On section, this is found to be due to the thickening of the coats of the vessel, and at this spot the vessel is blocked by an old clot which

has a cavity in the centre. Rest of basilar artery occupied by a recent soft black dot. No abnormality observed in other cerebral arteries. The ventricles of brain not distended, and interior normal to naked eye, except that the grey matter, especially in the motor areas, is extremely pale and almost white in places. This was most especially so in the left ascending frontal and parietal convolutions. Thorax: lungs distended, normal, except for some œdema at bases. Heart flabby, much post-mortem staining. Slight atheroma of aorta. No valvular disease. Very little clot.

Abdomen.—Liver normal; kidneys normal; spleen normal; intestines healthy; vermiform appendix situated behind cœcum, running downwards behind peritoneum on psoas muscle. Stomach, some p. m. staining and injection of vessels. No ulceration. No sign of gastritis or ulcer.

The above is a case in which general paralysis of the insane occurred in a man who has had syphilis in his early youth, and who had no trouble from secondary symptoms. In his case there was insane inheritance.

The earlier symptoms, which were at the time ascribed to general paralysis, passed off so completely that his father and his wife believed that he had recovered, and requested that he should be discharged "well." He was so well that for three months he performed his duties as chaplain to the satisfaction of his friends, and went alone to Greece for a holiday, where he broke down for the second time. I think the very unfavourable conditions in which he was placed in the Corfu Maison de Santé had much to do with the rapidity with which the disease developed.

On re-admission, he had general exaltation, and this continued till the time of the onset of vomiting, which in his case took the place of the earlier fits; this was followed by loss of consciousness and death, the respirations becoming very slow before the end.

At the post-mortem we found very characteristic specific changes in the vessels, as well as adhesions about the frontal lobes, and the grey matter of the motor tracts was very pale. The case seems to be one of ordinary general paralysis, due to arterial diseases, and answers a question which is frequently raised as to whether there is anything special in the symptoms of cases of general paralysis depending on syphilis, and the answer is "No;" but that in the course of the disease, if syphilis be the cause, remissions are more perfect than is usual with the cases depending on other causes.

I have made use freely of the notes taken by Dr. R. Percy Smith.

CASE OF HÆMORRHAGE INTO THE MEDULLA OBLONGATA—RECOVERY.

BY J. W. BATTERHAM, M.B., B.S. (LOND.), F.R.C.S.

E. L., a single woman, aged 33, had suffered for nine years from spasmodic asthma, attacks of which were of almost daily occurrence.

She came under my care in October 1886, with slight bronchial catarrh. Her heart's action was irregular and somewhat feeble. There was no cardiac murmur.

On October 26, 1886, the patient, during a fit of coughing, felt a severe pain in her head and down the left side of her body, and became giddy. She then noticed that her left arm was "very weak," and her speech indistinct. There were no convulsions, nor did the patient lose consciousness.

She remained under my care for about a fortnight, and exhibited the following symptoms—

Paresis of *left arm and leg*, more marked in the arm, the movements of the fingers being most affected.

Slighter loss of power in the *right arm*.

Marked impairment of the movements of the *lips*, especially on the *right* side, the power of movement of upper part of face remaining unaffected.

Deviation of the *tongue* to the *left*, when protruded.

Great indistinctness of speech, from defective movement of tongue and lips.

Dilatation of *left* pupil. Temporary difficulty of accommodation.

Paresis of *right* side of velum palati.

Difficulty of deglutition.

There was no anæsthesia, and no impairment of sight, smell, hearing (taste not tested), nor was there any paralysis of extrinsic muscles of the eyeball.

During the first few days of her illness the patient complained of constant nausea, and vomited occasionally. She frequently experienced sensations of "falling" and of "sinking through the bed."

Frontal headache was present for a few days. Ophthalmo-

scopic appearances were normal. The temperature was normal. Urine also normal.

The patient was removed to the Hastings and St. Leonard's Hospital, where, after a temporary increase in the severity of her symptoms, she recovered in about two months.

During the greater part of her illness she was *entirely free from asthma;* but, as she recovered from her paralysis, the asthmatic paroxysms gradually reappeared, and, on her discharge from the hospital, were as frequent as ever.

Remarks.—In 751 cases of cerebral hæmorrhage collected by Gintrac, the medulla oblongata was only *twice* found to be the seat of the lesion.

In support of the diagnosis in the present instance I would remark—

(1) As to the pathological diagnosis, that the suddenness of the attack was indicative of embolism or hæmorrhage, the former condition being improbable in the absence of cardiac valvular disease, while the fact that the onset occurred during a violent fit of coughing pointed strongly to the latter condition as the cause of the paralysis.

(2) As to the localisation of the lesion, that a hæmorrhage, the occurrence of which is unaccompanied by convulsions, loss of consciousness or fall of temperature, and from the effects of which the patient recovers completely in two months, must necessarily be limited in extent.

For a lesion limited in extent to produce paralysis of widespread distribution on both sides of the body, it must be situate in a part of the encephalon where the motor tracts of opposite sides are closely approximated. It must be either in the pons or medulla.

The special symptoms of *pontine* lesion, viz. those of injury to its nerve nuclei, were absent; while the symptoms which were present are for the most part readily explained by the hypothesis of *bulbar* injury.

The facial paresis noticed was such as might be produced by implication of the *accessory* nucleus of the seventh nerve, which is situated near the nucleus of the hypoglossal, and is supposed to be concerned chiefly with the movement of the lips in speech.

The ingual, palatine, and pharyngeal pareses are indicative of injury to the hypoglossal and spinal accessory nuclei; the nausea, vomiting, and, presumably, the remarkable cessation of asthmatic paroxysms point to an interference with the vagal nuclei. The dilatation of the left pupil and temporary indistinctness of vision may have been due to irritation of cilio-spinal fibres. The "giddy feelings," "sensations of

falling," "sinking through the bed," &c., are difficult of explanation unless we assume that the "inner auditory nucleus," with which the posterior root of auditory nerve is connected, has no auditory function, but receives those fibres of the auditory nerve which proceed from the vestibule, and which convey impressions giving rise, not to auditory perceptions, but to a "sense of direction." If such be the case, it is possible that an encroachment of the hæmorrhage on this so-called "auditory" nucleus may have originated the subjective sensations experienced by the patient.

I regret that I had no leisure to make anything like a complete investigation of this case, but trust that its rarity and its several points of interest may be an excuse for the publication of these scanty notes.

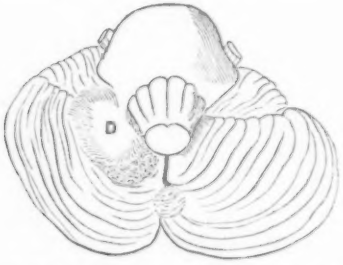
A CASE OF DEFECT IN DEVELOPMENT OF THE RIGHT LOBE OF THE CEREBELLUM, ASSOCIATED WITH DIMINUTION (?) OF THE OPPOSITE CEREBRAL HEMISPHERE.

BY PERCY FLEMMING, M.B., &c.

Demonstrator of Anatomy in University College, London.

THE brain, the subject of this note, was accidentally found in the dissecting-room, and in consequence there is no history attaching to it.

In viewing the base (see figure), a portion of the right hemisphere of the cerebellum is seen to be wanting, and the whole of that hemisphere is distinctly smaller than the left.



D. Defect where the white fibres are exposed by the failure of development of the laminæ.

More particularly, the deficiency is found to include almost the entire flocculus, the outer and anterior parts of the biventral lobe, and the adjacent part of the amygdala. The laminæ bounding the depression caused by the absence of these parts are all rounded off and covered by grey matter. In the floor of the depression is the white matter of the hemisphere, this being everywhere covered by pia mater.

The left cerebral hemisphere appeared markedly smaller than the right, but it only weighed half an ounce less.

However, in view of the fact that Sir J. Crichton-Browne ('BRAIN,' Vol. I., p. 504; II., p. 42) found, in opposition to

Dr. Boyd, that the average excess of the right over the left cerebral hemisphere was very small, this difference of half an ounce assumes more importance than appears at first sight. The difference in the apparent size of the hemispheres was also more marked than the weight would suggest; perhaps there was also a difference in the specific gravity, but this point could not be determined.

Reviews and Notices of Books.

Anleitung beim Studium des Baues der Nervösen Centralorgane im gesunden und kranken Zustande. By Dr. Heinrich Obersteiner, k. k. a. ö. Professor a. d. Universität zu Wien. Leipzig u. Wien: Toeplitz u. Deuticke, 1888.

We have great pleasure in bringing this work before the readers of "Brain," as we are confident that it will supply a want which is much felt, and it will not require any advocacy on our part to recommend it.

The book is of especial value, as it brings into juxtaposition the normal and pathological appearances of the minute structure of the nervous system, whereby the recognition of minute departures from health is much facilitated. The work is a pure study of the normal and minute pathological anatomy, and does not treat of the physiology or the clinical symptoms of diseases of the nervous system.

The book is divided into seven different sections.

In the first section, which treats of the methods of preparation of the various parts of the central nervous system, all the different modes of examining the tissue, such as teasing, hardening in the various reagents, the question of imbedding and cutting by various microtomes, are minutely discussed, and here we find Fol's modification of Fleming's method of staining the nuclei by perosmic acid and chromic acid, and a complete account of imbedding in celloidin.

Further on in this section the different ways of staining the nuclei, the medullated sheath and the axis-cylinder of the nerves are given, and especially Weigert's hæmatoxylin-copper method for staining the medullated fibres, and Pal's modification of the same, in which the hæmatoxylin is washed out with a solution of potassic permanganate, and one of oxalic acid and potassic sulphite; to this method Prof. Obersteiner gives the preference over the original one of Weigert, as it possesses the advantage of allowing the sections to be subsequently stained by carmine or eosine.

Weigert's method of fixing a series of sections between two layers of celloidin, and the corrosive sublimate process of Golgi, by which the various cells and their processes are so beautifully stained, are also minutely described.

Lastly, the other means by which the nervous system may be examined, viz. Flechsig's developmental method, Gudden's extir-

pation method, the mode of tracing the fibres by degeneration, caused by disease or operation, are discussed, as well as the evidence obtained from comparative anatomy and from experimental work.

It will be seen from the above that Professor Obersteiner has provided a very complete account of the various methods of examining the nervous system, including all the latest discoveries, and has thus furnished a concise reference, which will be invaluable to the practical worker in nervous histology.

The second section treats of the coarse anatomy of the nervous system, including the convolutions of the brain. This is illustrated by numerous original drawings, and we would call special attention to Figures 6 and 7, which are furnished with a numbered outline sketch to explain the various superficial parts of the posterior aspects of the medulla and pons. Here we would like to speak most highly of the way in which the whole work is illustrated. The drawings are nearly all original, and Professor Obersteiner has adopted the useful method of supplying an outlined numbered plan to accompany each illustration, instead of obscuring the original drawing with figures.

The third section treats of the histological elements of the nervous system.

Here, under the headings of nervous and non-nervous constituents, we have in the former list the nerve fibres and the nerve cells minutely described, as they are met with in health, and the condition in which they are found in disease; this includes the different stages of degeneration and regeneration of nerves, hypertrophied axis-cylinders, simple atrophy of nerve-cells, fatty and granular degeneration and calcification of ganglion cells, and the different conditions are illustrated by numerous drawings depicting the changes.

In the non-nervous constituents are included the vessels, epithelium lining cavities, and the supporting tissues, including connective tissue and neuroglia. In the description of the vessels, the perivascular and the pericellular lymph spaces are well shown, and Professor Obersteiner calls attention to the fact, that a lining membrane exists outside the adventitia of the cerebral arteries, from which numerous fine fibres radiate into the brain substance. He also remarks on the presence of pigment and fat granules on the normal adventitia of the small cerebral vessels, especially among children; but if the amount of fat granules be increased to a large extent, they must be looked upon as pathological.

The other changes in the vessels, such as calcification, changes in the intima, miliary aneurisms, and enlargement of the adventitial lymph space, are illustrated by original drawings, which show the actual condition better than can be described in words.

In the fourth section, in which the minute structure of the spinal cord is considered, the general arrangement of the connections of the different tracts of the cerebro-spinal system is detailed, and the different systems of Meynert, Flechsig, and Luys are enumerated. The different parts of the spinal cord are then

described as regards its topographical and histological structure. Prof. Obersteiner calls in question the propriety of describing the anterior commissure of the cord as a true commissure, as it probably contains fibres which are decussating, and very few which connect parts having a similar character. At the end of this section the acute and chronic affections of the spinal cord are described, as far as their pathological changes are concerned.

In the fifth section, on the topographical survey of the brain, we have presented a drawing of the medulla, pons, and lateral ventricles, as seen from behind and above. The region between the lower end of the medulla and the anterior border of the corpora quadrigemina is marked off in this drawing by eighteen transverse lines, and for each of these lines a corresponding representation is given of a transverse section of the pons and medulla. We have therefore a series of eighteen transverse sections, which show every change in structure in the pons and medulla. The drawings are magnified to four times the original size, and in the region of the crura cerebri, a whole page is devoted to the representation; one half of the drawing is a most artistic picture, while the other half is drawn in outline, and contains the letters explaining the different parts. This arrangement is of inestimable value in helping the investigator to determine where to make a section on the medulla or pons, to ascertain exactly the condition of any given nerve nucleus—a task which is often exceedingly difficult. All the sections being so perfectly treated, it is not easy to draw attention to any particular one; but we might cite the sections showing the facial nerve, as an instance of the clearness with which its very difficult course is demonstrated. Probably no man has a better knowledge of the most intricate parts of the medulla and pons than Prof. Obersteiner, and he has treated this subject in a most masterly manner.

At the end of this section the complicated parts of the subthalamic region are described.

In the sixth section the various principal fibre-tracts are traced between the brain and the spinal cord, comprising the pyramidal tract, the posterior columns of the cord and the tracts in connection with it—namely, the lemniscus, the restiform body, and the other tracts of the cord, including the one first described by Gowers. These systems are illustrated by two diagrams, containing the probable course of the fibres. The different cranial nerves are then described very fully, and diagrams given to explain the deep origin of the 1st, 2nd, 5th, 7th, and 8th nerves. These diagrams are most useful, and convey more to the student than any amount of printed description could attempt to do, and they show at a glance the deep origin of these nerves according to the views of the present time.

It is impossible to discuss the way in which the individual nerves are treated, but suffice it to say that all the latest discoveries are embodied in the work.

The article on the cerebellum is of course very complete, as Prof. Obersteiner has largely contributed to the elucidation of its

structure. We would call special attention to Fig. 151, which gives a representation of the protoplasmic processes of one of the Purkinje cells stained by Golgi's sublimate method, by which means an exceedingly dense and complicated network of fibres is seen to exist.

The structure and relation of the central grey masses of the brain are next discussed, including the optic thalamus with its complicated connections.

In the description of the corona radiata, amongst other things the fibres coming from the posterior part of the median convolutions and from the occipital convolution are described as going to the posterior part of the internal capsule, as well as to the thalamus, the outer corpus geniculatum and the anterior corpora quadrigemina.

The minute structure of the different convolutions of the cortex cerebri are next portrayed, and the fine network of medullated fibres of the cortex, as shown by Weigert's method, is illustrated by a drawing.

The work is concluded with sections on the membranes of the brain and cord and the large vessels.

It will be seen that this work is a most valuable contribution to the study of the anatomy and pathology of the nervous system, and we cannot speak too highly of the ability and skill which Prof. Obersteiner has brought to bear on this most difficult subject. We can confidently assert, that it will be an invaluable aid to all who are working at the pathology and anatomy of the nervous system, and we can only hope that its usefulness in this country will be further enhanced by its translation into English.

C. E. Beevor.

A Manual of the Diseases of the Nervous System.—By W. R. Gowers, M.D., F.R.S. 2 vols. 8vo, 463 and 975 pp. 370 figs. Churchill, London, 1886–1888.

The first instalment of this work, containing the Diseases of the Spinal Cord and Nerves, is no doubt already familiar to many of our readers; but we are glad that the publication of the second—more important, as well as voluminous—gives us an opportunity of noticing the work as a whole. We must, however, deny ourselves the pleasure of writing at any length concerning Dr. Gowers' volumes; for they belong to the category of works indispensable to those interested in Neurology, upon whose shelves they will speedily occupy a conspicuous position. Their contents, moreover, are so vast as to make it impossible in a review to enumerate the subjects handled by the author, far less to attempt an analysis and discussion of the views held by him on the numerous problems with which he has to deal. We shall limit ourselves, therefore, almost entirely to a statement of the leading features of this Manual, that characterise it as one of the very best published in any language.

What we admire, first, is the clearness of thought and language in the exposition, even in the more difficult portions of the subject. It is not every one who being a master, is at the same time a skilful expounder, and knows how to elucidate, whilst condensing, his theme. Secondly, we find the evidence, on every page of the book, of the author's individual familiarity with the topics he is discussing. Dr. Gowers' work in this respect often partakes of the interesting freshness of an original treatise, such is the wealth of personal information woven into his exposition of the subject in hand. Finally, we note the thorough mastery of the author of the most recent researches: a most creditable accomplishment when one considers the disproportion between the time at the disposal of a London physician in full practice, and the wealth of our contemporary neurological literature. Nor are the chapters devoted to the scientific or theoretical aspects of neuropathology the only ones deserving praise; those devoted to the practical aspect of the question, to nerve-therapeutics—this too often neglected "partie honteuse" of our branch of medicine—will be found if not always sanguine and cheerful, at least earnest and suggestive. Readers of French and German works on nervous diseases will however notice the larger share of attention given by Dr. Gowers, in common with most English authorities, to internal medication as compared with that bestowed upon hydrotherapeutics, massage, active and passive movements, and the like, under the various headings of treatment.

The Manual contains accounts not only of the leading morbid types, but of numerous minor forms of nerve disturbance, many of them to be found mentioned only in quite recent papers and monographs; and its interest and value are thereby enhanced for the professed neurologist, as much as for the student and general physician. Dr. Gowers would, however, add still further to our debt of gratitude to him as a guide through the field of neuropathology if, in his next edition, he were to give us a chapter of the pathology and treatment of stammering, a subject too much left to the "exploitage" of empirics. Morphinism might advantageously be adverted to under the heading of intoxications; and perhaps a sketch of the leading symptoms of general paralysis would be useful, especially in connection with certain phases of tabes. Another item which we miss in the present volumes is an index to the figures, many of which are very instructive. These suggestions, however, hardly deserve the name of criticisms. So in order not to derogate to the time-honoured tradition of reviewers always to find some fault with the work under notice, we shall attract Dr. Gowers' attention to some verbal mistakes in the course of the book. Thus, for instance, on p. 104 we find "Sepelli" for Seppili, "die" for "der" Complex; p. 924, the French expression for pleurosthotonos should be "arc de cercle latéral," &c. &c. But these are minor points. We find that Dr. Gowers adopts the absurd "pontine" for "pontal." Pontine is the name of marshes near Rome, but not the adjective derived from pons. Again, as the Greek "method" gives "methodic," so anŏd and kathŏd (usually

misspelt and mispronounced anōde and kathōde) give anŏdic and kathŏdic, not anōdal and kathōdal. Again "faradaic," is not only pedantic but inexact: in forming an adjective from the name Faraday, the terminal diphthong *ay* should be replaced by the termination *ic*. With reference to the expression *myotatic*, introduced to designate the nature of the phenomena of tendon excitability known as knee-jerk, &c., we think (after some hesitation, we must confess) that it is a useful one, and deserving to be generally adopted.

A. DE WATTEVILLE.

Critical Digest.

CHOREA IN THE ADULT, AND IN THE OLD.

BY W. P. HERRINGHAM.

CHOREA is not a common disease in persons after thirty years of age, and the records of it are scanty; but the term is used of so many different classes of illness, that it is worth while to arrange and classify the cases at present recorded.

In the first place, elderly or old people are occasionally, though very rarely, attacked by a chorea which in course, duration, and result cannot be distinguished from the disorder so common in the young. Thus Roger[1] has recorded a case in which an old lady of 83 was affected by an ordinary chorea which came on without cause, and was cured in five weeks; Russell[2] writes of a woman of 77, who had it mainly on the left side, and recovered in three months; Sinkler gives a case of a man of 86 who recovered in a few months; Saundby[3] gives two cases, but the histories do not extend for a sufficient length of time to enable them to be classified. Ferguson[4] had a case in a woman of 74, who after much fatigue in nursing her husband, became somewhat irritable and excitable, and then began to twitch on the left side. She recovered in eleven weeks. In the Collective Investigation Report,[5] there are several cases which Dr. Mackenzie has grouped together as chorea at exceptional ages. No. 119, by Gowans, is a second attack of chorea following rheumatic fever in a man of 40. No. 223, by Alexander, is a woman of 63, in whom the attack lasted four months. She also had had it before. No. 222, by the same author, is a first attack in a woman of 68, lasting six

[1] 'Gaz. d. Hôp.,' 1854, xxvii. 559.
[2] 'Med. T. and G.,' 1879, ii. 447.
[3] 'Lancet,' 1884, ii. 948.
[4] 'Lancet,' 1885, ii. 92.
[5] 'B. M. J.,' 1887, i. 425.

months. Carline records a case, No. 180, of an old woman of 73, in whom a chorea lasted four weeks. She had had several attacks of late years. Aitken describes another case, No. 93, in a woman of 86, lasting one year. Of this kind also is Graves's case[1] of a man of 70, in whom the attack lasted several months, and Gauthier[2] records an attack in a woman of 75, due to peripheral irritation from extraction of a tooth, similar to those occasionally met with in the young. She recovered after fourteen days.

This class of case is like the chorea of the young, in that it is curable, and lasts a comparatively short though variable time, from a week or two to several months. There is not, however, if we may judge from so few cases, the same tendency to heart disease, nor is rheumatism so frequent an antecedent. In Nos. 119 and 180 of the Collective Investigation Report, and in one of Dr. Saundby's cases, the heart was diseased; in two it is not mentioned; and in Roger's, Russell's, Ferguson's, Gauthier's, and the remaining Collective Investigation cases it is normal. Acute rheumatism had occurred in No. 119, is not mentioned in four cases, and had not occurred in any of the others.

In the next class comes the chorea of coarse sub-cortical lesion, which often follows hemiplegia, sometimes precedes it, and sometimes is without it altogether. Whether, however, there be hemiplegia or no, the class is distinguished by possessing a coarse lesion, deeper than the cortex, and by limitation of the movements to the other side of the body. The lesion is usually a patch of softening, either, as originally described by Charcot, in, or pressing on the posterior part of the internal capsule behind the motor tract, or, as in the case of Gowers,[3] and apparently in that of Galvagni,[4] entirely within the optic thalamus; or again, as in the case of Gee,[5] in front of the motor tract of the internal capsule. It appears to me probable that Tuckwell's cases of plugging of the posterior and middle cerebral,[6] so often, but I must think wrongly, quoted

[1] 'Clin. Med.,' i. 537.
[2] 'J. de Méd. et Chir. Prat.,' 1881, lii. 66.
[3] 'Brain,' Vol. I. p. 57.
[4] 'Riv. Clin. d. Bologna,' 1880, x. 268.
[5] 'St. Barth. Hosp. Rep.,' xvii. 285; and 'Brit. and For. Med. Chi. Rev.,' 1867.
[6] 'Brit. and For. Med. Chi. Rev.,' 1867.

in support of the theory which ascribes chorea to capillary embolism, are really to be placed in this class.

A third class is formed by a disease of which there are few notices, in which many members of a family are attacked by chorea in adult, or middle life, which lasts till death, and is usually accompanied by insanity. Huntington [1] was the first to describe such cases. I am unable to find his paper in London, and therefore rely on the account of it given by Wharton Sinkler.[2] Huntington himself, his father, and grandfather, knew some families in Long Island in whom this form had been under observation for many years. It came on gradually, after middle life, and affected males more than females. In most cases, insanity and a tendency to suicide supervened. Ewald[3] writes of a mother and two daughters in whom an incurable chorea began at or after thirty, and also of another family in which nine individuals of three generations were similarly afflicted. Peretti[4] gives a family tree, showing many individuals in three generations affected in middle life with chorea, a considerable proportion of whom were also insane. King[5] records another family, in three generations of which the disease has appeared, and gives a good description of the condition as he has observed it. These cases began about thirty years of age, were always associated with insanity, and showed a peculiar gait. West, in the Collective Investigation Report,[6] describes another family similarly affected as regards movements, but does not mention insanity in connection with it. There have been no post-mortems recorded of this disease.

The remaining or fourth class of cases differ from the first class by being chronic, and incurable; they differ from the second, in that when a lesion has been found, it has been such as to affect the cortex rather than the deeper parts; and they differ from the third in the absence of any hereditary tendency.

[1] 'Philad. Med. and Surg. Reporter,' 1872, Ap. 13.
[2] Art. "Chorea" in Pepper's 'System of Med.,' vol. 5, 440.
[3] 'Ztschft. f. Klin. Med.,' vii. Supplement H. p. 51.
[4] Peretti, 'Berl. Kl. Woch..' 1885, No. 52.
[5] 'N. Y. Med. Journ.,' 1885, i. 468.
[6] Loc. cit.

This class is divisible into two groups: (1) of cases preceded or accompanied by mania or dementia; (2) of cases with no mental alteration. Of the first group, Hale[1] records the history of a coachman, aged 65, who had chorea for six years, had two attacks of mania during that time, was very intemperate, very irritable, and died of acute bronchitis. He had cerebral pachymeningitis, and his brain was shrunken, very pale, and hard. Maclaren[2] relates the case of a man of 36, in whom the chorea had begun seven years before, after severe frontal headache. He became childish, and died. He had subarachnoid hæmorrhage over both hemispheres for a considerable area, and general atrophy of the convolutions, with a small spot of softening in the right optic thalamus. The movements were general, not confined to the left side, and therefore probably not to be explained by the disease of the thalamus. Mackenzie Bacon[3] describes a woman of 58, in whom the chorea lasted four years, and was combined with mania. Dissection showed chronic arachnitis, a shrunken brain, and a large collection of serum in the ventricles. Macleod[4] gives three cases. The first is that of a man aged 52, in whom chorea lasted six years; dementia ensued, and he died in coma. He had a subdural hæmorrhagic cyst over both motor areas, and the convolutions were atrophied. The second is a woman aged 62, who had chorea for six years, gradually became demented, and had a similar subdural cyst over the left motor area, with a thick and adherent pia mater. The movements were general. The third is her sister, aged 72, who died in stupor after three years' chorea, and who had several hard tumours in the dura mater. Devis[5] describes an old man of 79 with dementia, who, while in hospital for a broken thigh, developed left-sided chorea and died, but was not dissected. Berkley[6] gives a long and careful account of the microscopic examination in the case of a woman of 41, who seven years ago developed chorea five hours after being severely frightened by a murder committed before her. She gradually became demented, and died. He found in the brain

[1] 'Path. Trans.,' v. 16.
[2] 'Journ. Ment. Sci.,' xx. 97.
[3] 'Journ. Ment. Sci.,' xxvi. 253.
[4] 'Journ. Ment. Sci.,' xxvii. 194.
[5] 'Med. T. and G.,' 1879, ii. 446.
[6] 'Philad. Med. News,' xliii. 1883, 200.

"dilatation and thickening of arterioles, small gangrenous spots round many vessels; amyloid corpuscles in the lymph sheath; vacuoles in white and grey matter; varicosities of the sheath of Schwann, and absence of axis-cylinders in the nerve fibres; and complete pigmentary degeneration of a large number of the cortical and ganglionic cells, with loss of the prolongations." There were similar changes in the cord, the cellular degeneration being especially marked in the enlargements, while there was considerable antero-lateral sclerosis in the remainder of the cord. The heart was fatty, but without a trace of past or present endocarditis. Bevan Lewis [1] relates the case of a man, aged 56, who had general choreic movements after a paraplegia of the legs six years ago, and who had some mania six months before death. There was congestion of the meninges of the brain, with a granular degeneration throughout the motor cells of the cord, but the cortex of both brain and cerebellum was quite normal to the microscope, and there was nothing amiss in the basal ganglia.

It is of interest to compare with this group Case No. 365 of the Collective Investigation Report, also by Macleod, in which a woman, aged 40, was seized with a severe chorea immediately after blows on the head. There was mania for a month, and the chorea lasted five months. Macleod suggests a lesion in the front of the brain, evidently likening it to the fatal cases he had before recorded.

Of the second group, that in which no dementia or insanity exists, Russell [2] describes a case which had lasted over eighteen months, and was unrelieved by treatment, the patient being a man of 68. Sinkler [3] mentions another in a woman of 82. McLearn [4] relates a third in a man of 56, in which there was the subjective feeling of heat so common in paralysis agitans. None of these died under observation. Dickinson [5] had a case in a woman of 54, who died of granular kidneys. Nothing was found post mortem to explain the chorea. I had a case among my out-patients at the West London Hospital in a man of 68, who had had the disease for fifteen years. It affected

[1] 'Journ. Ment. Sci.,' xxx. 256.
[2] 'Med. T. and G.,' 1878, ii. 627.
[3] Loc. cit.
[4] 'Lancet,' 1885, i. 337.
[5] 'Med. Chi. Trans.,' lix. 1.

only the right arm and the head, which was continually being jerked to the right side, that is, to the side affected. Although so confined, it was violent. A continually increasing anæmia attacked him, for which he was admitted into the hospital under Dr. Donald Hood, and of which he died. I examined the brain carefully, and could find no abnormality in it whatever, but I regret to say that I did not make a microscopic examination.

A remarkable case is related by Wood,[1] in which a man of 74 suffered amputation of the left thigh for senile gangrene, and five years later began to be affected with irregular clonic spasm, mingled with some tonic spasm, called by Wood "chorea of the stump," which afterwards spread to the arm and face of the same side. There was never any pain connected with the chorea, nor did any kind of peripheral irritation increase it.

Charcot, speaking of the two groups of this class, remarks that in the few cases he has dissected, there has been no endocarditis, and that there is no connection with rheumatism. This is borne out by the other observers who have examined cases after death. I have mislaid the post-mortem notes of my case, but I do not remember any heart disease being found, and there was no murmur during life. The man had never had any previous illness.

It is worth noting that the only cases in which gross disease has been found have been in the insane. That these, however, are not constant in the brain, even in this group, is shown by the results of Bevan Lewis's examination. It is too early to lay any stress upon the changes hitherto recorded. All we can do is to classify the cases according to their clinical symptoms. To fulfil this task is the object of the present paper.

[1] 'Philad. Med. Times,' Nov. 3, 1879, quoted in 'Med. T. and G.,' 1880, i. 16.

Abstracts of British and Foreign Journals.

Pitres et Vaillard on Acute Ascending Paralysis (*Arch. de Physiol. Norm. et Path.*, 1887, p. 149).—The case was one of a man, æt. 22, convalescent from typhoid fever. He woke up one morning and found that his legs were paralysed and anæsthetic, with loss of reflexes. During the day the paralysis spread upwards, involving the abdominal muscles; later on, the thorax and arms were paralysed. Respiration became diaphragmatic, aided by the shoulder and neck muscles. Bladder and rectum paralysed. Deglutition not affected. Death in about 12 hours.

Post-mortem.—The central nervous system was quite free from disease. The nerves of the upper and lower extremities showed signs of disease as follows:

(*a*) Fibres atrophied to such an extent as to show only their primitive sheath with *débris* stained slightly by the osmic acid.

(*b*) Fibres in which the myeline was finely broken up and gathered into varicosities.

(*c*) Fibres in which the myeline was divided into larger masses. The axis cylinder was affected in a similar manner.

This description applies to such nerves as the post-tibial, sciatic, musculo-cutaneous, radial, &c. The spinal roots were affected to a much less degree.

The authors consider that this case throws some light on that mysterious disease described and named after Landry. They discuss the opinions of other writers at some length, and show that while a few have described change in the cord, a considerable number have shown that the central nervous system is free from disease, and several have described changes in the peripheral nerves.

Letulle on Mercurial Paralysis (*Arch. de Physiol. Norm. et Path.*, 1887, p. 301).—The author begins by noticing that the attention of most writers is directed chiefly to the tremors of mercurialism, and that the pareses have been passed over by the larger number of them. He shows that paresis, general or partial, is exceedingly common, and may be almost the only symptom. Anæsthesia and

even patches of hyperanæsthesia are very common. Pain in the affected limbs is rare. There is never any atrophy of muscles. The reflexes are increased in cases in which there is much tremor, but diminished often in the paretic cases.

The upper limbs are much more often affected than the lower. The trunk is rarely affected. He draws his cases from the mines of Almaden, and from the felt-makers in Paris.

But in order to study the effect of the poison on the nerves he subjected rats and guinea-pigs to as long a course of mercurial treatment as possible. His experiments were of three classes. (1) Injection of mercurial peptone along the course of a nerve. (2) Injection of the same anywhere in the body. (3) Inhalation of mercurial vapours.

In most cases he got a definite affection of the muscular nerves. This affection was apparently confined to the myeline sheath of the nerves, and therefore differs considerably from the ordinary parenchymate neuritis. It consisted of (1) swelling granulation of the myeline; (2) granular disintegration, followed latterly by (3) atrophy of the myeline. This condition obviously differs from other forms of neuritis, in the fact that only the myeline sheath is affected, the axis cylinder being spared indefinitely. This will explain the great rarity of atrophy in hydrargyrism, but it is difficult to explain the paresis, at least by the ordinarily accepted views of the functions of the myeline sheath.

The paper is well illustrated by cases and details of experiments.

H. H. Tooth, M.D.

Chauffard on Sudden Blindness in Lesions of the two Occipital Lobes (*Revue de Médecine*, Feb. 1888).—It is not easy to refer sensory functions to their place in the cerebral cortex, for not only are there few clinical facts to guide us, but experiments on the lower animals are open to obvious difficulties. The localisation of sensory thus affords a great contrast to the localisation of motor functions, which indeed may almost be regarded as a finished work.

In respect to Sight, it has been proved by Allen, Starr, Seguin, and Exner, that bilateral corresponding *hemianopsia* results, without any other sensory or motor symptom, from destruction of part of the occipital lobe. Cases have also been recorded in which in General Paralysis *total blindness* has been noted corresponding with lesions of both occipital lobes. Pflüger relates that a young man received the discharge of a gun in the occipital region, causing immediate blindness and ultimately double optic neuritis.

He died, and at the autopsy grains of lead were found scattered in both occipital lobes.

The author gives four cases, three of which are taken from Berger and Bouveret, in which sudden complete blindness was associated with lesions of these lobes. In his own case, that of an old man of seventy-four, there were three hæmorrhages, one, on the right side, occupying the greater part of the occipital lobe; and two in the left hemisphere. One of these lay in the external capsule, but the larger clot was contiguous to the *pulvinar*, at the very point where, according to Wernicke and others, the fasciculi of the optic tract leave the central ganglia. From this point they pass outside the hinder limb of the lateral ventricle, and finally reach that portion of the cortex which forms the cuneiform lobe and first occipital convolution. In the three other cases, also old men, the occipital regions were affected by softening, from thrombus or embolism. This association of symptoms and lesions accords well with the experimental results obtained in animals by Ferrier and Yeo.

Dr. Chauffard considers that in his case, as in others, simple hemianopsia must have preceded the total loss of sight. Hemianopsia may be often latent or undiscovered. Blindness is thus the result of two seizures on two separate occasions. After the first seizure (hæmorrhage in the left hemisphere) the uninjured centre doubtless took on by substitution more or less of the functions of the other. After the second hæmorrhage (in the right hemisphere), substitution became impossible. It is quite exceptional for the two occipital lobes to be attacked at once. The question arises whether hemianopsia, due to cortical lesion, tends by extension to the companion centre to pass into anopsia. If so, the symptom is one of serious import; for, according to the present author, cases of cortical anopsia invariably prove fatal within a short time. We may note, finally, as diagnostic features, that the blindness is complete and sudden, that "optical memory" is retained, that the fundi are normal, and, what is important, that the pupils are equal, moderately *dilated* (not contracted as in uræmic intoxication), and preserve their reflex excitability to light.

Spillman and Parisot on Peripheral Injury and Tabes (*Revue de Médecine*, March 1888).—In answer to the enquiries, "Can peripheral injuries be the starting-point of Tabes? Are they a sufficient cause?" the authors relate a series of cases in which locomotor ataxy followed the infliction of various injuries. It is not difficult to believe *à priori* that even a small lesion of a

part of the sensory system might lead to an affection generalised over the whole of that system; although it is impossible to assert without evidence from morbid anatomy, that is not at present forthcoming, *how* that generalisation is effected. But, in their opinion, clinical evidence establishes the fact.

The interval between the injury and the first symptoms of tabes varied from a few weeks to as much as eighteen years; and it is worthy of note that, in five out of the fourteen cases quoted, nervous heredity, alcoholism or syphilis were stated to be present. Former writers have noticed an occasional connection between injuries and locomotor ataxy; and Erb (quoting Schulze) cites cases in which it has followed fracture of the thigh or a fall on the abdomen. Petit considers that wounds at a distance "hasten its development," and others have counted *injuries* in a general way as among the causes of tabes. The present writers carry this view much further. Given a personal or hereditary predisposition to nervous affection, they hold that, besides fractures, many slight injuries, such as sprains or wounds of the feet, or even operations in dentistry or for cataract, may be sufficient determining causes. Again, the first symptom of the tabes arises always, in their view, in the injured region. After injury to the left foot, the symptoms commenced after five months with lightning pains in the same limb; after contusion of the abdomen, with gastric crises, two months from the date of injury. This circumstance will often afford a guide in the history of each case to the original exciting cause. Finally it is allowed that diatheses—alcoholism, syphilis or rheumatism, may exert in some of these cases a predisposing influence. FORTESCUE FOX, M.D.

Raymond on Sweating of the Face (*Arch. de Neurol.*, Jan. 1888).—The first case recorded is that of a man, aged 31, who noticed in 1880 that sweating of the right side of the face always appeared two or three minutes after he had taken food, and ceased when he left off eating. The sweating extended on to the neck and as far as the shoulder. Hot food induced it most rapidly; movements and the idea of eating had no effect; all other symptoms were absent, except that the right pupil was dilated and not affected by light, and below the right wrist there was difficulty in distinguishing between hot and cold objects. Injections of two milligrammes of pilocarpin produced a local sweating of the face on the right. Neither galvanism, nor atropine, nor ergotine, nor tannin produced any effect on the patient's malady.

The author divides the cases of local sweating into the following

groups. First:—Those in which there is an alteration in the cerebro-spinal nervous system. Second:—Those in which the cervical sympathetic, or the first thoracic ganglion is affected. As a rule, in these cases, the pupil is contracted, but one is cited in which it was dilated. In this case there was chronic inflammation of the superior cervical ganglion on the same side. The author's second case is placed in this second group; it is that of a man, aged 39, who had phthisis; he also had unilateral right-sided sweating, especially of the forehead. There were no vaso-motor or pupillary symptoms. He died suddenly, three days after an epileptiform attack. The tubercular pleurisy had bound down and caused chronic inflammatory change in the right inferior cervical ganglion. Third:—Those in which the nerves of the face are affected. Fourth:—Those in which the sweating is reflex. These cases usually occur in nervous hysterical people. Many examples of this group are given, such as that of a young man in whom facial sweating was induced by smelling vinegar.

Thus there are two classes of cases characterised by increased sweating; those in which there are and those in which there are not vaso-motor troubles. The lesion in the former is in the sympathetic of the neck; the fact that the pupil is sometimes dilated and sometimes contracted depends upon the fact, that the pupillary and vaso-motor nerves are probably distinct, and one set may be stimulated whilst the other is paralysed. The author then gives an account of the various chronic inflammatory changes that have been found in the superior cervical ganglion, and concludes that these irritate the sweat-secreting nerves. Sometimes these changes exist without there being any alteration of sweating. Lastly, he points out, that the pupillary changes are permanent whilst the sweat ones are transitory; that the increased secretion of sweat may extend beyond the face, so that treatment has very little effect.

HALE WHITE, M.D.